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STUDIES IN EXPERIMENTAL TRAUMATIC SHOCK

IV. EVIDENCE OF A TOXIC FACTOR IN WOUND SHOCK*

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During the last four years, clinical observations and laboratory studies have added materially to our knowledge of wound shock. Summarizing old and new features of this condition it may be said that it is characterized by a low venous pressure,[1] a low or falling arterial pressure, a rapid thready pulse, a diminished blood volume,[2] a normal or increased erythrocyte count and hemoglobin percentage in peripheral blood,[3] a leukocytosis,[4] an increased blood nitrogen,[5] a reduced blood alkali,[6] a lowered metabolism,[7] a subnormal temperature, a cold skin, moist with sweat, a pallid or grayish or slightly cyanotic appearance, by thirst, by rapid respiration, often by vomiting and restlessness, and by anxiety, changing to mental dulness and lessened sensitivity. Many of these features may appear at once or as soon after the reception of the wound as the observations can be made; or they may develop only after the lapse of several hours.

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* Previous articles in this series are: Aub, J. C.: I. The Basal Metabolism, Am. J. Physiol. **54**:388 (Dec.) 1920. Aub, J. C., and Cunningham, T. D.: II. The Oxygen Content of the Blood, Ibid. **54**:408 (Dec.) 1920. Aub, J. C., and Wu, H.: III. Chemical Changes in the Blood, Ibid. **54**:416 (Dec.) 1920.

1. Wiggers, C. J.: Am. J. Physiol. **46**:326 (June) 1918. Erlanger, J.; Gesell, R., and Gasser, H. S.: Ibid. **49**:106 (June) 1919.

2. Keith: Blood Volume in Wound Shock, English Medical Research Committee, Report Series No. 26, March 14, 1919, p. 36. Gasser, H. S.; Erlanger, J., and Meek, W. J.: Am. J. Physiol. **50**:31 (Oct.) 1919.

3. Cannon, W. B.; Fraser, John, and Hooper, A. N.: Some Alterations in Distribution and Character of Blood in Shock and Hemorrhage, J. A. M. A. **70**:527 (Feb. 23) 1918.

4. Govaerts: Ambulance de l'Océan **2**:359, 1917. Keith: Blood Volume in Wound Shock, p. 8.

5. Duval and Grigaut: Compt. rend. Soc. de biol. **71**:873, 1918. Aub, J. C., and Wu, H.: Am. J. Physiol. **54**:416 (Dec.) 1920.

6. Cannon, W. B.: Acidosis in Cases of Shock, Hemorrhage and Gas Infection, J. A. M. A. **70**:531 (Feb. 23) 1918.

7. Aub, J. C.: Am. J. Physiol. **54**:388 (Dec.) 1920.

INITIATING FACTORS IN SHOCK

The onset of early or *primary* shock is most reasonably accounted for as a consequence of some disturbance of the nervous system. A review of the evidence at hand shows that it is impossible to eliminate as a consequence of wounds a reflex relaxation of blood vessels similar to that which occurs in fainting. Indeed, as Cowell [8] has observed, fainting is, in fact, not infrequently observed after the reception of wounds. Vincent [9] likewise has seen cases of this character; but in the only instance which he describes in detail (that of a man wounded in the abdomen, who, a few minutes after being hit, manifested the syndrome of shock), the blood pressure, at the end of forty-five minutes, had risen from 60 to 90 millimeters of mercury. It is possible, then, that there may be produced by a wound an effect similar in character to fainting or syncope, but persisting for a longer period than the usual fainting spell.

British, French and American observers had occasion during the war to note that shock may begin to appear two or three hours after the wounding. It may be associated with continued bleeding, but commonly cannot be explained by hemorrhage or infection. Exposure to cold, lack of water, rough transportation, and the agitation of broken bones have been recognized as circumstances favorable to its development. These factors may later have their action increased by infection, especially by the bacilli of gas gangrene. This is *secondary* wound shock and was by far the most common form noted during the war. An analysis of the theories which have been most commonly advocated in the past, such as inhibition,[10] reduction of the carbon dioxid content of the blood (acapnia [11]), fat embolism,[12] exhaustion of nerve centers and of certain glands,[13] shows that they do not offer an adequate explanation for the onset of delayed or secondary shock. Their chief and common defect is that they fail to account for the occurrence, both in clinical and in experimental shock, of a diminution of blood volume and either a local or a general concentration of blood corpuscles. A group of theories which do take these facts into consideration, namely, those which postulate a primary vasoconstriction with a consequent capillary congestion,[14] fail in that they do not suggest

8. Cowell, E. M.: The Initiation of Wound Shock, J. A. M. A. **70**:608 (March 2) 1918.

9. Vincent: Compt. rend. Soc. de biol. **81**:887, 1918.

10. Meltzer, S. J.: The Nature of Shock, Arch. Int. Med. **1**:578 (July 15) 1908.

11. Henderson: Am. J. Physiol. **21**:128, 1908.

12. Porter: Harvey Lectures, Philadelphia, 1917-1919, p. 21.

13. Crile, G. W., and Lower, W. E.: Surgical Shock and the Shockless Operation Through Anoci-Association, Philadelphia, W. B. Saunders Company, 1920.

14. Malcolm: Tr. Med. Soc., London **32**:274, 1909. Starling, E. H.: Arch. méd. belges **71**:369 (Oct.) 1918. Erlanger, J.; Gessell, R., et al.: Am. J. Physiol. **49**:90, 151, 345, 1919; Ibid. **50**:31, 104, 119, 1919.

how a vasoconstriction would occur, capable of bringing about a reduction of blood volume. What is required is a demonstration of some factor, naturally related to the production of shock, which may so operate in the body that, when hemorrhage and infection are ruled out, the persistent low blood pressure characteristic of shock will become gradually established.

CHARACTER OF INJURIES ASSOCIATED WITH WOUND SHOCK

A primary consideration in experimental study of natural phenomena is that the phenomena reproduced under controllable conditions shall resemble as closely as possible those occurring in nature. This is a consideration which seems to have been largely neglected in experimental investigations of shock. No doubt, persistent low blood pressure can be produced by a variety of means. In support of various theories of shock, investigators have been able to duplicate certain aspects of the condition by protracted stimulation of nerves, by severe and long-continued manipulation of exposed intestines, by vigorous over-ventilation of the lungs, by repeated pushes against the stomach, by interrupting the circulation in large areas of the body, or by injecting into the blood stream relatively large amounts of oil or cream. Although such procedures cause changes similar to those which occur in natural shock, they do not reproduce the circumstances which occasion shock after ordinary wounds or injuries. Obviously, it is desirable to duplicate, so far as possible, the actual conditions which give rise to a shocklike state, and then to analyze them in order to determine what factor, or factors, among them is operative.

The typical nature of the wounds seen in cases of secondary shock may be judged from the following records made by a group working at Béthune.[15] In reading these records, it should be understood that the wounds are often made by large ragged pieces of shell moving at high velocity. When such a missile breaks the femur or the ilium, for example, it smashes and tears extensively the overlying muscles, which, in the thigh and hip, form a thick layer. In all these cases, shock was present:

CASE 1.—E. G., compound fracture of left ulna and radius, arm nearly severed, wounds of right arm and left side, abdomen opened, intestine and omentum protruding.

CASE 2.—C. P. H., shell wounds of right thigh with fracture of the femur, and shell wounds of left arm with fracture of the humerus, wounds of the face.

CASE 3.—G. D., wounds of the left arm with fracture, left leg with fracture, flesh wounds of the right thigh and abdomen.

CASE 4.—G. J. H. H., shell wounds of right ankle, of left leg with fracture, and of the muscles of the buttock.

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15. Cannon, W. B.: Acidosis in Cases of Shock, English Medical Research Committee, Report Series No. 25, Dec. 25, 1917, pp. 86-88.

CASE 5.—H. J. H., bursting shell broke right femur, left tibia and fibula, and injured right arm.

CASE 6.—A. J. R., extensive wounds of both legs, left foot and left arm.

CASE 7.—W. G., wounds of buttock with fracture of ilium, also wounds of right foot.

CASE 8.—P. K., fracture of left femur (much comminuted) and right tibia.

CASE 9.—W. A. T., wounds of buttock and perineum, muscles below the buttock torn across and smashed in both legs.

CASE 10.—G. K., wounds of both arms, left thigh, left foot, compound fracture of the right thigh.

CASE 11.—R. C., compound fracture of right femur, multiple shell wounds, left femur, buttock and chest.

CASE 12.—A. H., multiple shell wounds; large wound of the left loin involving the gluteal muscles, fracture of the pelvis, extensive wound of right calf muscles and the muscles of the left thigh and left calf, and numerous wounds in back and chest.

The general character of war wounds is well described by Wallace and Fraser:[16]

> The tissues of the wound are crushed and lacerated, and there are widespread contusion and effusion of blood into the surrounding parts. The neighboring blood vessels are often pulped and thrombosed, and as a result of the interference with the blood supply whole areas of tissue may afterward die and slough away. In these wounds, muscle appears to be affected more than any other tissue; it becomes a mass of dark brown crushed matter without any evidence of striation or vitality. One cannot fail to be impressed by the enormous destruction which even a small fragment of shell will produce, a degree of destruction which is apparently quite out of keeping with the size of the fragment; the exaggerated damage depends on the enormous velocity at which the fragments are traveling.

EXPERIMENTAL EVIDENCE OF A TOXIC ORIGIN OF SECONDARY WOUND SHOCK

After spending the summer of 1917 at a casualty clearing station in Béthune, I went to London and worked for three months with Bayliss in an experimental study of shock. At first, we were interested in the question as to whether the acid known to be developed in injured tissue might not be a contributory factor in the production of shock as seen in man. The traumatization which we caused induced both a reduction of blood alkali (an acidosis) and a low blood pressure (Fig. 1). Later it was shown that the acid production was not an essential factor in the complex.[17] Some other agent, therefore, must be acting. Further analysis of the mode of operation of the trauma will be deferred until the observed facts are described. These facts were estab-

16. Wallace and Fraser: Surgery at a Casualty Clearing Station, London, 1918, p. 31.

17. Acidosis and Shock, English Medical Research Committee, Report Series No. 25, October, 1919, p. 250.

lished in part by Bayliss and myself working together,[18] and in part by each of us working independently, after I was transferred to the Laboratory of Surgical Research of the American Expeditionary Forces at Dijon, France.

In order to bring about, in lower animals, a traumatization similar to that giving rise to shock in man, the thigh muscles in the anesthetized cat, while being supported by an iron block, were repeatedly struck with a blunt wedge-shaped hammer, or they were crushed by compression. The trauma usually failed to break the skin, so that infection from without was impossible. Occasionally, the femur was broken, but this had no influence on the results which were seen. After this procedure, the course of events was followed in observations on the pulse, respiration and the alkali reserve and the corpuscular volume of the blood. In Figure 1 is presented a summary of the changes seen in a typical case. An actual record of the blood pressure changes of shock produced in this way is shown in Figure 2.

As Figure 2 proves, the crushing of the muscles in one hind leg of the cat may have no immediate effect. There may be a momentary rise or fall of arterial pressure. After about twenty minutes, however (and still longer in the dog), the pressure begins to fall. And after about an hour, the pressure has usually fallen to 80 millimeters of mercury, or even lower, i. e., to a shock level. There it may persist for several hours. In other words, the general bodily condition resembling shock is produced by duplicating the circumstances which induce shock in man. The only noteworthy difference is the presence of anesthesia (urethane). That this is not a determining factor is shown by the use of a controllable anesthetic, such as ether, which may be lightened as the animal falls into deeper and deeper insensibility. The lessening of the ether concentration under these circumstances does not improve the circulation.

The fall of pressure after traumatization might be regarded as due to loss of blood and lymph into the damaged tissues, for there is always considerable swelling of the injured region. This suggestion was tested, however, by removing *postmortem*, with symmetrical cuts, the two hind legs, one normal, the other injured, and weighing them. The difference of weight which, in some instances, was only 10 per cent.

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18. For early reports of these observations, see: Cannon, W. B.: Med. Bull. Med. Research Soc., Am. Red Cross, Paris, April, 1918, p. 426; Comot. rend. Soc. de biol. **81**:850, 1918. Bayliss, W. M.: Intravenous Injection in Wound Shock, New York, Longmans, Green & Co., 1918. Cannon, W. B., and Bayliss, W. M.: Note on Muscle Injury in Relation to Shock, Report of a Special Committee on Surgical Shock, English Medical Research Committee, March 14, 1919, p. 19. Bayliss, W. M.: Further Observations on the Results of Muscle Injury and Their Treatment, Ibid., p. 23. Cannon, W. B.: Some Characteristics of Shock Induced by Tissue Injury, Ibid., p. 27; Mil. Surgeon **44**:494 (May) 1919.

of the estimated blood volume, would not represent enough extravasated blood to account for the fall of pressure. It must be admitted, of course, that loss of blood by extravasation, even when slight, may play a rôle in the subsequent development of the low pressure.

The widespread effect in the organism induced by the trauma might be due to nervous impulses. Such impulses on passing to the central

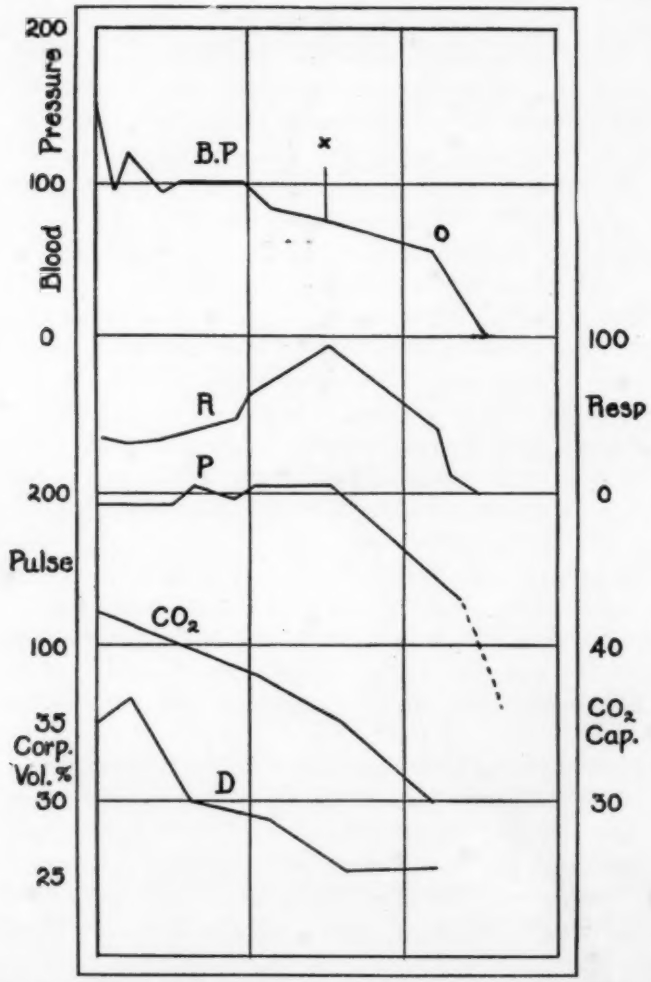

Fig. 1.—Record of changes in the blood pressure, respiration, pulse and carbon dioxid capacity (alkali reserve) of the plasma after injury of the flexor muscles in the thigh; urethane anesthesia. At *X*, asphyxia caused the rise of blood pressure indicated; at *O*, no rise in asphyxia.

nervous system might so affect it as to produce reflex inhibition of the vasoconstrictor center, as has been suggested above for the initiation of primary wound shock. Is such the case? The nature of the experiment permits securing evidence on this point. It is only necessary to

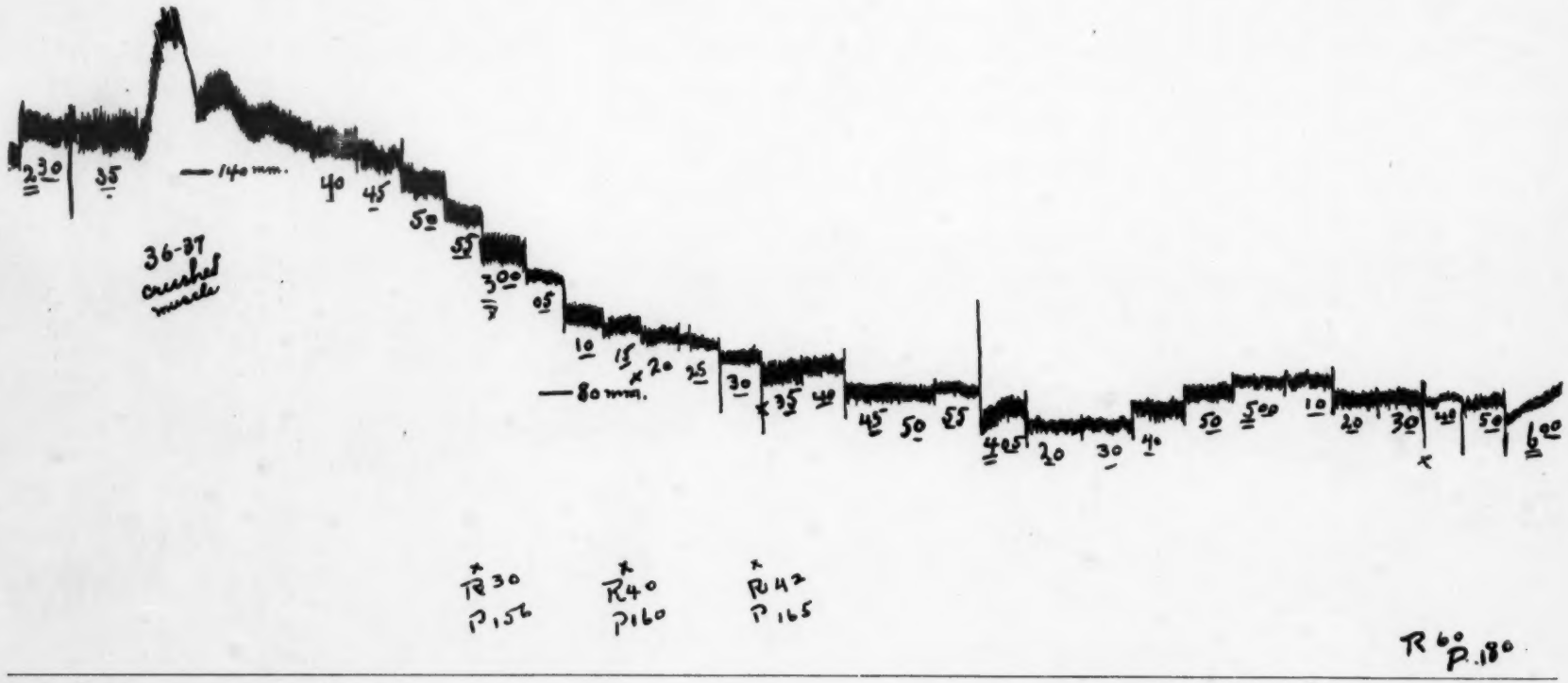

Fig. 2.—Typical fall of arterial blood pressure after crushing muscles of the thigh (at 2:36-2:37): *R*, respiration; *P*, pulse.

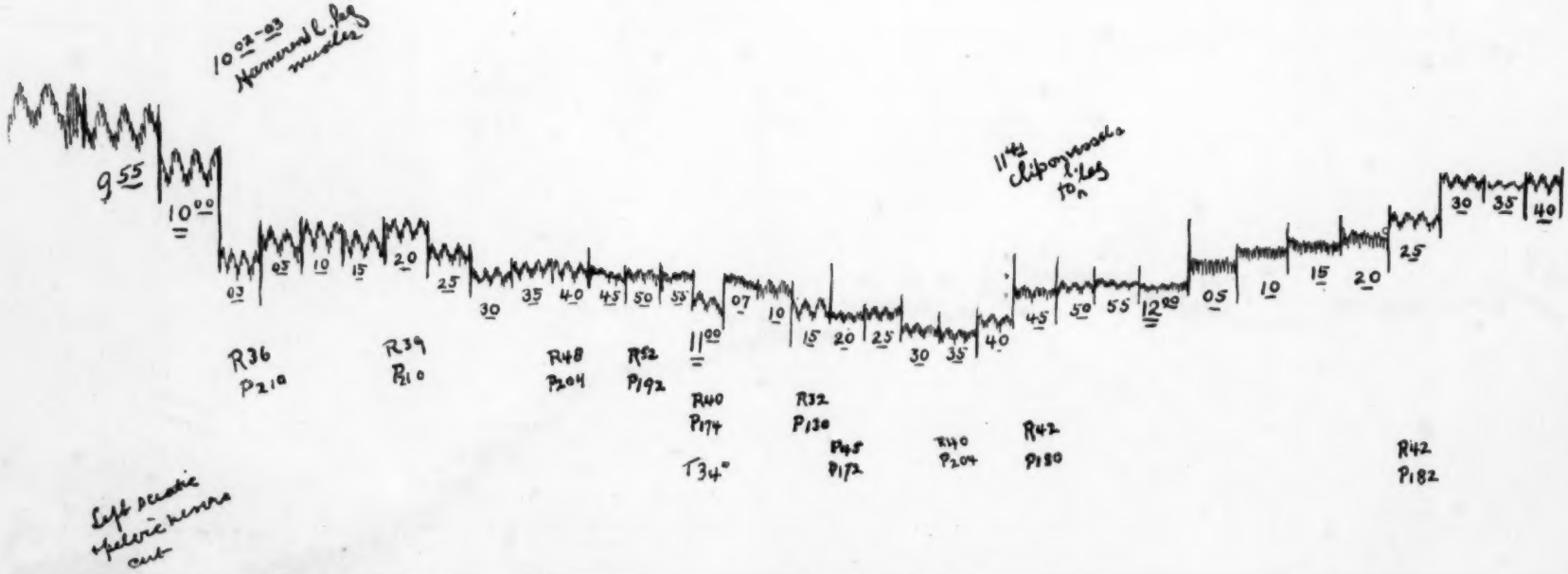

Fig. 3.—Fall of blood pressure after muscle injury (10:02-10:03), though the nerves to the injured limb had been cut; rise of blood pressure to the original level after closing (at 11:41) the blood vessels to and from the injured region: *R*, respiration; *P*, pulse.

transect the spinal cord above the lumbar plexus or to sever all nerves of the limb which is to be injured, in order to disconnect the region from the central nervous system. When this has been done and the denervated muscles are traumatized, events occur similar to those seen after trauma when the nerves are intact (Fig. 3). From this evidence it is clear that there is no essential relation between the production of shock and an excessive stimulation of the central nervous system.

The gradual fall of blood pressure, again, might be interpreted as due to depression or exhaustion of the central nervous system, with gradual loss of vasoconstrictor tone. That this is not true was shown by tests performed by Cattell in the Harvard laboratory. He used the method described by Bartlett, in which the rate of inflow of a fluid at constant pressure through the femoral artery of one hind limb was determined

SUMMARY OF RESULTS SHOWING PERFUSION TIME FOR 1 C.C. OF PHYSIOLOGIC SODIUM CHLORID SOLUTION IN RELATION TO BLOOD PRESSURE IN MUSCLE INJURY SHOCK

Experiment	Control Perfusion Time, Seconds	Maximal Perfusion Time, Seconds	Period After Injury, Hours	Percentage Increase	Perfusion Time at End, Seconds	Perfusion Time After Death, Seconds	Original Blood Pressure, Mm.	Blood Pressure at Maximal Constriction, Mm.
8	5.2	12.9	0.75	144	4.7	...	140	90
9	2.3	3.3	1.0	43	2.9	1.8	120	16
10	1.4	2.4	4.0	71	...	1.2	140	74
12	1.2	2.7	4.5	125	1.1	0.8	130	68
14	0.8	1.5	1.5	87	1.0	0.5	130	60
15	1.3	3.7	1.25	185	...	1.3	120	72
16	1.6	4.0	3.5	150	...	...	135	78
18	2.7	4.0	4.5	48	...	...	125	65
19	1.5	2.5	6.0	66	...	1.1	135	42
20	1.4	2.5	...	78	...	...	130	92
21	1.0	1.7	3.0	70	0.9	0.6	130	60
Average	1.85	3.74	3.0	102	2.1	1.0	131	65

at intervals during the development of shock produced by injury of the other hind limb. The inflow cannula was always placed in a side branch of the femoral artery, so that when injections were not being made, a clip on the main trunk could be removed to permit the leg to receive its natural blood supply. In Cattell's tests, about 3 c.c. of physiologic sodium chlorid solution was allowed to run into the artery while the time was being recorded with a stop watch. In a few instances in which perfusion was interfered with by small clots forming in the vessels, the experiments were discarded. The condition of the vessels was checked by inflow determinations after death, when, if there is no obstruction, the rate will be greatly increased. In the accompanying table is a summary of the results. The figures represent the time in seconds for the entrance of 1 c.c. of fluid, and in each case they are the average for at least three determinations. Invariably, there was a gradual increase in the time (i e., decrease in the rate) of inflow after muscle injury, usually starting within the first hour and reaching a

maximum in from two to four hours. At this time of maximum constriction, the blood pressure had already fallen to a shock level, the average pressure for the series of experiments being 65 millimeters of mercury. After this, a dilation occurred which continued until death and was accompanied by a further fall of blood pressure. As already stated, the perfusion rate was still further increased after death. A curve showing the general relation between the blood pressure and peripheral response is given in Figure 4, which illustrates the averages of six experiments which were carried on over a period of four hours or more. From these results, it is apparent that the low pressure initiated by muscle injury is not primarily due to a loss of vasomotor tone or to a dilation of the blood vessels. In other words, this form of shock is not the consequence of any form of exhaustion. This conclusion is similar to that reached by Erlanger, Gesell and Gasser for shock produced by manipulation of the intestines and by occlusion of the aorta.

It might be supposed, further, that the local injury, especially when the femur is broken, would permit a sufficient amount of fat to be liberated to cause the pressure to fall. In several instances, careful examination of the lungs was made by expert pathologists, Louis B. Wilson and Henry W. Cattell; but in no instance was there evidence of any accumulation of fat. Furthermore, there was no twitching or disturbance of respiration or other sign which might indicate that fat had passed through the pulmonary capillaries into the central nervous system. The fall of pressure, therefore, cannot be attributed to fat embolism.

Although there was usually a change of respiration in the direction of greater rapidity and lessened amplitude, this was not characteristic of the period immediately after the trauma, but developed as the blood pressure fell. Commonly, the respiration became slower toward the end (Fig. 1). The absence of hyperpnea for some time after the injury, although the carbon dioxid capacity of the plasma was falling, and the later rapid and shallow breathing, which, since it was superficial, was not such as to induce a washing out of carbon dioxid from the blood, rule out acapnia as a cause of the lowered blood pressure. This conclusion was confirmed by producing shock by muscle injury, though the breathing was kept uniform by artificial means.

If the low blood pressure resulting from local trauma is not due to loss of blood into the injured region, or to reflex vasodilation, or to depression or exhaustion of the vasoconstrictor center, or to fat emboli, or to acapnia, the connection between the local damage and the general bodily state may reasonably be looked for in the remaining great connecting system—the circulation. There may be given off from the damaged tissue into the flowing blood or lymph material, which, when

carried to the rest of the body, proves toxic and disturbs the control of the walls of the vessels in such a way as to lower arterial pressure. This idea can readily be tested. The blood vessels of the leg (the iliac artery and vein) are tied and the muscles are then crushed. In the experiment recorded in Figure 5, the blood vessels of the leg were tied before the muscles were smashed, and the ligatures were left in place for thirty-three minutes after the trauma. The record shows that there was no drop of blood pressure during this period, although the arterial pressure usually begins to fall in the cat about twenty minutes after the injury. As soon as the blood flow was restored, however, the pressure promptly fell to a low level. This phenomenon can be explained

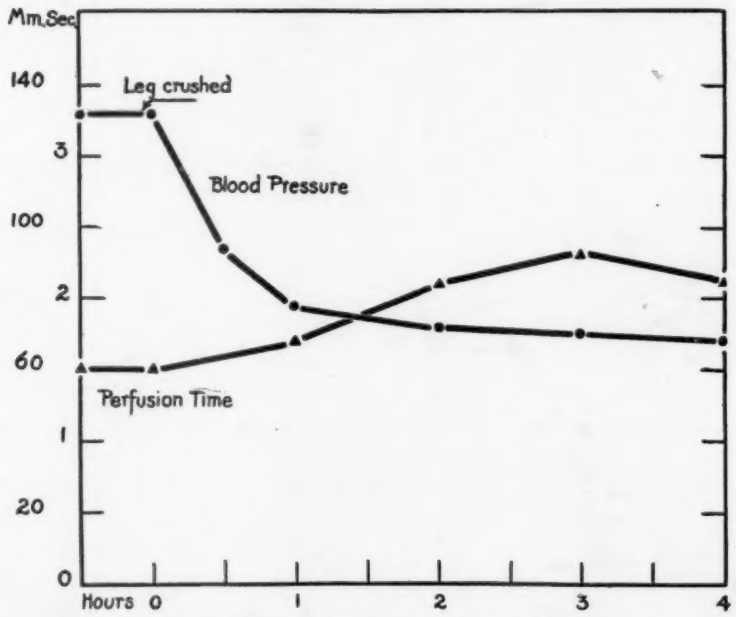

Fig. 4.—Curve plotted from the averages of six experiments showing the relationship between the perfusion rate and the blood pressure in muscle injury shock. As the blood pressure falls, there is a slowing of the perfusion rate indicating an increased tone of the arterioles.

on the supposition that when the blood is again allowed to flow a pressure-lowering substance passes, by way of the circulation, from the traumatized region to the rest of the body. The inference just set forth received support from experiments in which the muscle was injured; and, while the pressure in consequence was falling, the vessels of the leg (the iliac artery and vein) were closed. As shown in Figure 3, occlusion of the vessels may be followed by a progressive rise of pressure to the normal level. If the injury is not great, the blood pressure, after falling for a while, may spontaneously be restored. The

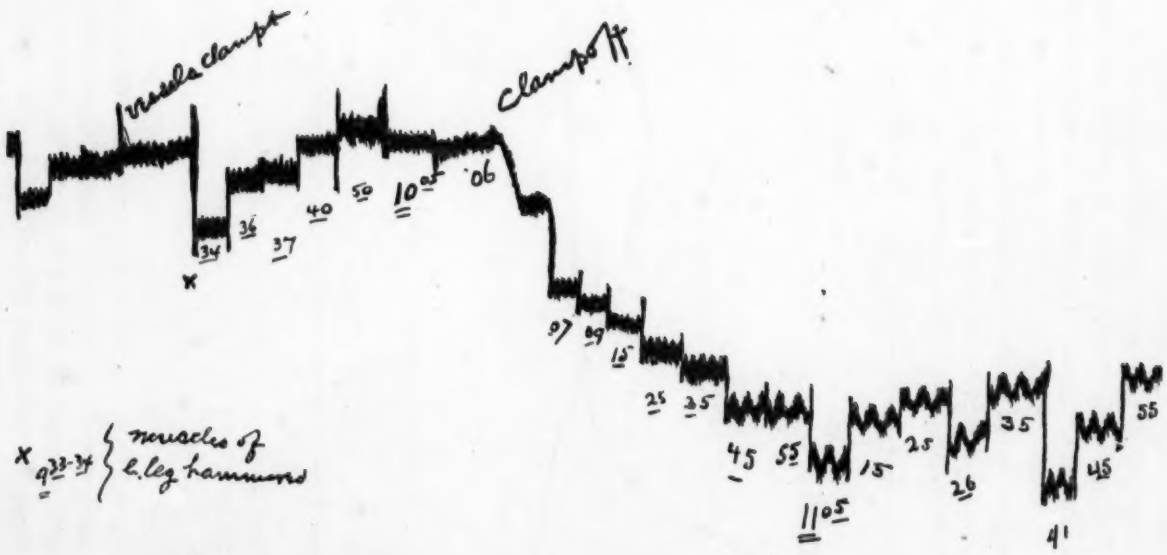

Fig. 5.—Failure of blood pressure to fall at the usual time after muscle injury (at 9:33-9:34) if the blood vessels to the injured region have been tied; on restoration of the blood flow (at 10:06), the pressure promptly falls.

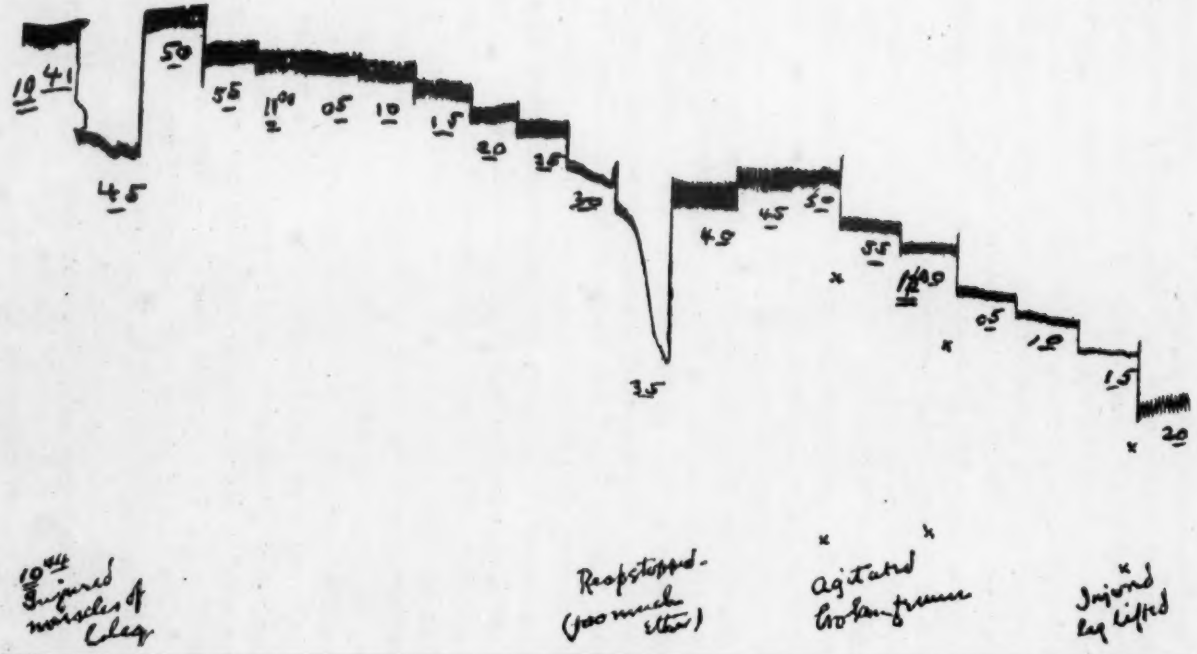

Fig. 6.—Increased rate of pressure drop after muscle injury when the ends of the broken femur were moved in the damaged tissues at *x* and *x*.

evidence indicates, therefore, that whatever may be the substance originating in the damaged tissues, it is fairly promptly changed in the body or eliminated, so that its effect is not permanent.

Further confirmation of the inference that a pressure-lowering material is given off from injured tissue was obtained by massage of the traumatized region. The result was a further drop in pressure. In Figure 6 is presented the record of blood pressure as it was falling after a smashing of the thigh muscles with fracture of the femur. At the points marked *x*, the fragments of the bone were moved in the damaged soft parts. The records taken shortly thereafter showed a greater drop than usual. The practical bearing of these observations on the importance of immobilizing bony fragments is obvious.

Other experimental work has led to the same conclusion that Bayliss and I reached in our experiments. In a recent publication, Turck [19] has declared that the consequences of cell necrosis brought about in various ways affect the whole organism in such a manner as to give rise to some of the symptoms of shock. In June, 1918, he reported experiments [20] on injury to the thigh muscles, that were similar to those performed by Bayliss and myself in January, and referred to in March and April of that year.[21] Unfortunately, his experiments were not accompanied by blood pressure records; and commonly, the injection of what he called "shock-toxins" was followed by rapid death of the animal rather than by the production of shock. In November, 1917, Delbet,[22] impressed by the similarity between traumatic shock and the shocklike conditions accompanying peritonitis, raised the question whether intoxication did not play a rôle in certain forms of traumatic shock, whether shock was not, in fact, the result of absorption of toxins arising from disorganized and bruised tissues. Delbet and Karajonopoulos,[23] in July, 1918, reported the effects of injecting into guinea-pigs and rats the products of autolysis of crushed muscles. They found that these substances were highly toxic, sometimes making animals comatose a few seconds after intraperitoneal injection. Respiration was accelerated; the reaction to noise disappeared; the animal became immobile, and in a variable time died—effects attributed by Delbet to disturbances of the nervous system. These experiments revealed a toxic material in muscle autolysates; but it did not produce detailed analogies to the state of shock in human beings. Delbet's view that toxic substances arise from autolytic disintegration of

19. Turck: Med. Rec. **95**:472 (March 22) 1919.

20. Turck: Med. Rec. **93**:927 (June 1) 1918.

21. Bayliss, W. M.: J. Physiol. **52**:17, 1918. Cannon, W. B.: Med. Bull., Med. Research Soc., Am. Red Cross, Paris, April, 1918, p. 426.

22. Delbet: Bull. et mém. Soc. de chir. **43**:2121, 1917.

23. Delbet, P., and Karajonopoulos: Bull. Acad. de méd. **80**:14 (July 2) 1918.

muscles was questioned by Vallée and Bazy,[24] who noted that autolytic ferments of muscles are slow in action. They expressed doubt whether autolysis would result in a toxin sufficiently abundant or active to induce shock, and suggested that bacteria and possibly leukocytes might induce proteolytic changes capable of hastening the production of poisonous material.[25] It seems unnecessary to assume that tissue disintegration should be carried far in order to evoke, when injected, a fall of blood pressure. As long ago as 1903, Vincent and Sheen [26] proved that watery extracts of a variety of tissues, when injected into the blood stream would cause a drop of arterial tension, and Perret,[27] in 1909, proved that fluid pressed from raw meat ("myoserum") was highly toxic when injected intravenously.

AN EXPLANATION OF TRAUMATIC TOXEMIA

Explanation of the shock-producing effect of substances which may arise from injured tissue has its beginnings in classic experiments in the history of physiology. Nearly thirty years ago Heidenhain [28] demonstrated that the injection of "peptone" into the circulation would cause an increased production of lymph, persistent low blood pressure and a notable concentration of the blood. These are changes which Starling,[29] in 1894, attributed to increased capillary permeability, especially in the liver.

In 1919, Dale and Laidlaw [30] called attention to a reduction of blood pressure which can be induced by the injection of extremely minute amounts (1 or 2 mg. per kilogram in the anesthetized animal) of histamin, a substance derived from the amino-acid histidin by removal of carbon dioxid. Later, attention was called to the shocklike character of the changes which histamin occasions.[31] The arteries are constricted (the early constriction of arteries in shock may be due, in part, to the action of toxic substances), there is oligemia, a reversed ratio of corpuscles to plasma, and failure of the cardiac output.[32] If the chest is opened, the heart is seen beating regularly; but its chambers contain little blood.[33] The arteries and veins likewise contain little blood.

24. Vallée and Bazy: Bull. et mém. Soc. de chir. **44**:707, 1918.
25. Vallée and Bazy: Bull. et mém. Soc. de chir. **44**:708, 1918.
26. Vincent and Sheen: J. Physiol. **29**:254, 1903.
27. Perret: Trav. du lab. de Richet **6**:91, 1909.
28. Heidenhain: Arch. d. f. ges. Physiol. **49**:252-254, 1891.
29. Starling: J. Physiol. **17**:39, 1894.
30. Dale and Laidlaw: J. Physiol. **61**:318, 1910.
31. Dale and Laidlaw: Memorandum Upon Surgical Shock and Some Allied Conditions, English Medical Research Committee, February, 1917.
32. Dale, Laidlaw and Richards: Traumatic Toxemia, English Medical Research Committee, Report Series No. 26, March, 1919, p. 9.
33. Dale, Laidlaw and Richards: Traumatic Toxemia, p. 10.

Moreover, by the use of vital red, Dale [34] demonstrated that a considerable part of the blood passes out of currency. It appears, therefore, that since the blood is not in arteries and veins, it must be concentrated in capillary areas. Dale reports that the bowel shows a diffuse dusky congestion and that the smallest venules are rendered visible on its surface by their content of dark blood—a condition similar to that observed by Erlanger and his collaborators [14] in the various forms of shock which they produced. By direct microscopic examination after the injection of histamin, Hooker [35] and also Rich [36] have shown that a large number of capillaries, previously not seen, become clearly defined because dilated and filled with blood. Dale and Richards [37] have further analyzed the action of histamin. The low pressure resulting from it is not due to relaxation of the arterioles. It is apparently due to a series of changes in which dilation of the capillaries and pooling of blood within them, poisoning of their endothelial walls so that they are abnormally permeable, escape of plasma through these walls into the tissue spaces, and consequent concentration of the corpuscles [38] are the main features. This thickening of the blood would accentuate the tendency for it to gather in the small vessels.

The great significance of these observations by Dale, Laidlaw and Richards is that the action of histamin may reasonably be regarded as typifying the action of a large class of poisonous protein derivatives—products of partial digestion, of bacterial action, and of tissue extraction. In 1909, Popielski [39] called attention to the depressor action of organ extracts and showed that "vasodilatins" could be extracted from practically all tissues. In 1911, Barger and Dale [40] proved that histamin was present in the mucosa of the small intestine, in Popielski's "vasodilatins," and also in commercial peptone. Recently, Able and Kubota [41] have adduced evidence that histamin is a widely distributed constituent of animal tissues, organ extracts and enzymatic products, whether derived from animal or vegetable proteins. In all probability, histamin is not the only constituent of tissue extracts and tissue digests capable of lowering blood pressure. Hanke and Koessler,[42] for example, have shown that typical peptone shock can be produced by injecting peptone from which histamin is absent. We may follow Dale, therefore, in

34. Dale, Laidlaw and Richards: Traumatic Toxemia, p. 11.
35. Hooker, D. R.: Am. J. Physiol. **54**:49 (Nov.) 1920.
36. Rich: J. Exper. Med. **33**:287 (Feb.) 1921.
37. Dale and Richards: J. Physiol. **52**:144, 1918-1919.
38. Dale, Laidlaw and Richards: Traumatic Toxemia, p. 12.
39. Popielski: Arch. f. d. ges. Physiol. **128**:191, 1909.
40. Barger and Dale: J. Physiol. **41**:499, 1911.
41. Abel, J. J., and Kubota, S.: J. Pharm. & Exper. Therap. **12**:297 (June) 1919.
42. Hanke, M. T., and Koessler, K. K.: J. Biol. Chem. **43**:567 (Sept.) 1920.

regarding histamin as a type of the toxic substances derived from protein material. Although proof is still lacking that a substance, like histamin in character, is actually given off into the blood stream when the tissues are severely damaged, the effects of local tissue injury (influencing the rest of the body solely through the circulation) and the effects induced by histamin are so similar that the supposition has a high degree of probability. In both conditions, there is a fall of blood pressure. In both, there is a reduction of blood volume. In both, there is, at least in the early stages, a concentration of the blood (Fig. 1). In both, slight hemorrhage results in markedly increasing the shocklike effect. It is a matter of considerable interest that proteolytic bacteria, such as those producing gas gangrene, induce, in muscles, chemical changes, which, according to Zunz,[43] are accompanied by the production of histamin. Extracts of such muscles are highly toxic and rapidly depress the circulation.[44] The association of "shock" with gas infection can thus be accounted for.

The view that secondary shock is due to action of toxins arising in injured tissue places it, as Dale [45] has pointed out, in the same general category with anaphylactic shock, for that likewise is probably the consequence of poisoning by protein cleavage products. The shocklike condition produced by toxic substances arising from intestinal obstruction which Whipple and his collaborators [46] have done so much to elucidate, becomes also closely related to traumatic shock, for chemical study led them to the conclusion that the poisonous agent developed in a closed intestinal loop belongs to the primary proteoses. It is conceivable, furthermore, that the results of methods used by other investigators to produce shock, such as pinching the intestine, as employed by Githins, Kleiner, Meyer and Meltzer,[47] or interfering with the circulation, as practiced by Janeway and Ewing [48] and by Erlanger and his group,[49] may be accounted for on the basis of a toxin production. Bayliss and I [18] found that after the blood flow had been wholly shut out from the hind limbs for an hour, restoration of the circulation resulted in a sharp fall of blood pressure. And Dale [32] proved that if the blood vessels of the fore limb are rendered sensitive to the action of histamin by denervation, letting the blood flow through the hind

43. Zunz: Compt. rend. Soc. de biol. **82**:1079, 1919.

44. Report of the Shock Committee, English Medical Research Committee, Report Series No. 25, October, 1918, p. 265.

45. Dale: Proc. Roy. Soc., B **91**:133, 1920.

46. Whipple et al.: J. Exper. M. **17**:286, 307, 1913; Ibid. **19**:144, 166, 1914; Ibid. **23**:123, 1916.

47. Githins, Kleiner, Meyer and Meltzer: Proc. Soc. Exper. Biol. & Med. **16**:3, 1918.

48. Janeway and Ewing: Ann. Surg. **59**:158, 1914.

49. Erlanger, J., and Gasser, H. S.: Ann. Surg. **19**:389 (April) 1919.

limbs after it had been shut out for some minutes is followed not only by dilation of the vessels of the hind limbs, but also, as in histamin injection, by dilation of the vessels of the sensitized fore limb as well. Since changes in the direction of death and cell disintegration are induced when the circulation is cut off from a region—changes which are accelerated by trauma— the suggestion seems reasonable that the methods mentioned above, used by other investigators to cause shock, likewise involved toxin production.

It should not be supposed that smashed muscle is the sole source of toxic material. Pinching the intestine, as stated above, will induce shock; and I have found that crushing the tips of two or three lobes of the liver may be effective also. Possibly, substances given off by extravasated blood are likewise depressive to the circulation; at least, the injection of blood just previous to its coagulation causes a sharp drop of blood pressure.

CLINICAL EVIDENCE FOR TRAUMATIC TOXEMIA

Aside from the experimental evidence which has resulted in the building up of a theory of traumatic toxemia as a cause of shock, there are also clinical observations which extended over approximately the same period as the experimental studies, and which, quite independently, led to the same conclusion. For the clinical studies which developed and gave support to the theory of toxemia as a cause of secondary wound shock, great credit should be awarded to Quénu, professor of clinical surgery in the Medical Faculty of the University of Paris. As early as 1916, having repeatedly noted in his military service the relation between the occurrence of shock and the attendant gross damage of tissue, he began to suggest to various surgeons in the French army the desirability of securing clinical data regarding the possible presence of a toxic factor in the production of traumatic shock. In December, 1917, the observations of Rouhier, one of his former interns, were reported by Quénu; and during 1918, a series of papers were presented to the Société de chirurgie in Paris by Quénu and others—Moulinier, Santy, Marquis, La Croix, Bertein and Nimier, Rouhier and Soubeyran—which brought striking confirmation of the ideas which had been expressed. Since an excellent summary of these papers has been made by Quénu,[50] it will be unnecessary to report them here in detail. For present purposes, a brief outline of the clinical evidence that secondary shock is a traumatic toxemia will suffice.

1. Secondary shock, or what the French call "shock primitif," does not appear immediately after the receipt of wounds, as a large number of observers have testified; and therefore it is not of the nature of a

50. Quénu: Rev. de chir. **56**:204, 1918.

nervous effect. Furthermore, the state is commonly well established before infection; and therefore it is not of bacterial origin.

2. Secondary shock is characteristically observed in association with extensive damage of muscles or with multiple wounds [51] scattered over the body. This contention of Quénu is supported by the studies at Béthune and by the observations of McNee, Sladden and McCartney,[52] who described cases in which, without injury to bones or to any vital structure or organ, there was extensive laceration of muscle, accompanied by such severe shock that most of the patients died in spite of treatment. In the case of multiple wounds the large number of small areas of damage would obviously be in sum the equivalent of destruction of a considerable mass in one region. The increase of undetermined nitrogen in the urine and of total nonprotein nitrogen, as well as residual nitrogen, in the blood of shock patients [5] may be accounted for as a consequence of the absorption of material from the traumatized area, and also, perhaps, as the effect of tissue damage done by circulating toxins.

3. Everything that favors absorption at the region of injury is favorable to the development of shock. Thus, in the observations reported by Quénu,[51] the development of shock is most severe when the region of damage communicates with the exterior by only a small orifice. The negative aspect of this evidence is presented by cases in which a large fleshy mass, along with the skin which covers it, is carried away by a missile; in such instances, shock is slight or wholly absent.[53]

4. Anything that delays or checks absorption from the injured region delays the development of shock; but if there is a sudden romoval of the check, serious results follow. In support of this point, Quénu [54] cites an incident observed by Rouhier:

> A man with a crushed foot was brought in with a tourniquet placed very tightly below the knee. The leg was tense, dark-colored and full of blood; but there was no sign of shock. Rouhier performed a conservative operation and then removed the tourniquet. Three hours after the operation, the patient was in a state of intense shock.
>
> Gregoire [55] has reported the analogous case of a lieutenant caught in a dug-out after a shell burst. His left thigh was compressed between two logs. Thus, he remained for twenty-four hours, alert, guiding the efforts of those

51. Quénu: Rev. de chir. **56**:270, 1918.

52. McNee, Sladden and McCartney: Observations on Wound Shock, Especially with Regard to Damage of Muscle, English Medical Research Committee, Report Series No. 26, March, 1919, p. 35.

53. Quénu: Rev. de chir. **56**:271, 1918.

54. Quénu: Rev. de chir. **56**:272, 1918.

55. Gregoire, cited by Quénu: Rev. de chir. **56**:272, 1918.

who were delivering him. His general condition was good; but he was pale, with a pulse small and rapid and a slightly accelerated respiration. There was no wound; the foot, leg and knee, however, were purplish and cold; above the knee there were two deep hollows formed by the pressure of the logs. Some hours after his rescue, the officer became restless; and, although treatment for shock was undertaken, he died thirty-two hours after the pressure was removed from the leg. There was no indication of nervous depression and no bleeding in this case. The shock appeared on permitting the circulation to return to the damaged tissue.

Five cases similar to these were reported to me from various surgeons in the hospitals of the American Expeditionary Forces in France. One instance was especially striking: A tourniquet had been in place on the upper arm for an uncertain period; but the wound in the wrist was so slight that the surgeon proceeded to clean it and take off the compression. The patient was in good condition before the tourniquet was removed. A short time thereafter, however, he went into profound shock and died. It is obvious that the state developed in these human cases was quite like that developed experimentally by Janeway and Ewing, by Erlanger and his collaborators, and by Bayliss and myself, when a continued low blood pressure was induced by interference for several hours with the circulation to a considerable part of the body.

The converse of the foregoing observations is seen when after a very severe wound the region is isolated by a tourniquet and the limb is amputated proximal to, and without removing, the tourniquet. Rouhier [56] reports a case of this type in which no shock appeared either before or after the operation.

5. Suppression of the injured region, if not too long delayed, causes shock to disappear. Much evidence in support of this statement might be cited. I had occasion to note such cases at Béthune; and Quénu and Rouhier [57] have described in detail instances of the rapid disappearance of shock after cutting away traumatized tissue. McNee, Sladden and McCartney [52] have declared that when a quick amputation of a lacerated region is possible, "the operation is commonly followed by a remarkable and maintained improvement, so rapid and striking as to appear a direct sequel to the removal of the damaged limb." And they cite the case of a wounded man who was too shocked to allow any movement whatever, and in whom a similar good result was obtained by tying a tourniquet as tightly as possible around the limb which was so badly smashed that amputation was seen to be inevitable if the patient survived. The observations of Santy [58] on the effects of

56. Rouhier, cited by Quénu: Rev. de chir. **56**:272, 1918.
57. Quénu and Rouhier: Rev. de chir. **56**:273, 1918.
58. Santy, cited by Quénu: Rev. de chir. **56**:233, 234, 1918.

delaying operation on the severely wounded confirm on a larger scale the foregoing individual instances. In a series of seventy-nine cases of nontransportable wounded soldiers, in which the time between the receipt of the wounds and the surgical treatment was known, the mortality was only 11 per cent. when the operation was performed in the first three hours. It rose to 37 per cent. when there was a delay of between three and six hours (though infection is not marked until after six hours), and was 75 per cent. in the eighth and ninth hours. These figures vividly illustrate what may reasonably be expected if time is given, both for the disintegration of damaged tissue and the absorption of toxic material.

The foregoing experimental and clinical testimony, most of which was gathered only in the last two years of the war, gives a quite new turn to ideas regarding the nature of shock. In this connection, the argument of Groeningen,[59] written in 1885 in support of the nervous origin of shock, is of interest. He pointed out that shock is more frequent after shell wounds and crushing accidents than after cuts and stabs; more frequent with large missiles than with rifle bullets; more frequent with amputations and resections near the trunk than distant from it; more frequent in operations which expose a large rather than a small surface, and more frequent with a prolonged operation than with a short one. All these considerations, he cited as showing that violent stimulation and shattering of nerves are the occasion for shock. It is clear, however, that they may quite as well be instanced as the conditions which are most favorable for causing the death of considerable masses or areas of tissue (thus liberating toxic agents) and for rendering the organism sensitive to the action of such agents.

In the development of our conception of the effects of extensive burns, there has been an evolution of ideas similar to that which has occurred with regard to traumatic shock. Forty years ago, Sonnenberg [60] attributed death from burns to a reflex depression of vasomotor tone. Modern studies [61] have shown that there is, as in shock, a great increase in the number of erythrocytes, i. e., a concentration of the blood and an enormous mobilization of leukocytes. The suggestion of recent writers [62] is that here too, death, when delayed, is the outcome

59. Groeningen: Ueber den Shock, Wiesbaden, 1885.

60. Sonnenberg: Deutsch. Ztschr. f. Chir. **9**:138, 1877; Virchows Arch. f. path. Anat. **80**:381, 1880.

61. Becker and Schnitz: Klinische und chemische Beiträge zur Pathologie der Verbrennung, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **31**:416, 1919. Weiskotten, H. G.: Histopathology of Superficial Burns, J. A. M. A. **72**:259 (Jan. 25) 1919.

62. Bardeen: Bull. Johns Hopkins Hosp. **9**:137, 1898. Eyff: Centralbl. f. d. Grenzgeb. d. Med. u. Chir. **4**:428, 1901. Pfeiffer: Virchows Arch. f. path. Anat. **180**:367, 1905. Vogt: Ztschr. Exper. Path. u. Pharm. **11**:191, 1912.

of an intoxication, probably by protein derivatives set free from the area of tissue destruction. The present conception seems to be that not only the shock following burns, but also the delayed shock consequent on severe trauma, is properly placed in the same category with other forms of general depression of bodily functions and defective circulation due to the setting free of toxic material in the body.

FURTHER OBSERVATIONS ON THE LYMPHATIC ORIGIN OF CHOLECYSTITIS, CHOLEDOCHITIS AND THE ASSOCIATED PANCREATITIS*

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The most common conceptions of the pathogenesis of infections of the gallbladder and bile tracts are based on four assumed possibilities: (1) descending infection from the liver by bacteria carried down in the bile, (2) ascending infections from the duodenum up the common bile duct, (3) hematogenous infections of the gallbladder and ducts, and (4) a spreading infection through the wall of the gallbladder from an inflamed contiguous organ. Of these possibilities, it is only the last two which take into serious consideration the actual infection of the deeper layers of the wall of the gallbladder, despite the fact that microscopic examinations of gallbladders removed at operation, at least those which we have studied, demonstrate that there are inflammatory changes not merely in the mucosa but also in the deeper layers and that usually these changes are more pronounced in the deeper layers. The other two possibilities are concerned only with the entrance of organisms into the lumen of the gallbladder, and they do not explain their entrance into its walls. They assume also that contact infection of the mucosa of the gallbladder occurs easily and frequently. But there is no direct evidence to support the idea that cholecystitis occurs, except perhaps occasionally, merely as a result of the entrance of bacteria into the lumen of the gallbladder; and there is much evidence, both clinical and experimental, that such infections are probably rare.

This evidence consists of a number of facts: (1) In experiments on ten dogs in which large amounts of pathogenic colon bacilli were injected into the normal gallbladder, no cholecystitis was produced sufficient to be demonstrable by gross appearance or clinical signs, except in one case. In order to produce it with any degree of regularity, it was necessary either to obstruct the outflow from the gallbladder by ligation of the cystic duct or to injure the blood supply by ligation of the cystic artery. In this connection a statement by Meyer, Neilson and Feusier[1] is of interest. These authors state:

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* Part of this paper was read before the American Surgical Association, Toronto, June 15, 1921.

* From the Department of Surgery, Washington University School of Medicine.

1. Meyer, K. F.; Neilson, N. M., and Feusier, M. L.: The Mechanism of Gallbladder Infections in Laboratory Animals, J. Infec. Dis. **28**:456 (May-June) 1921.

It is generally known from the studies of Cushing and from our own, that typhoid bacilli introduced directly into the cystic bile of dogs disappear rapidly and that a cholecystitis can only be produced by considerable injury of the wall, or by placing a foreign body in the gallbladder (Marxer).

(2) Bacteria probably gain access to the gallbladder by being carried down in the bile from the liver in most, if not in all, cases of bacteriemia; yet cholecystitis is comparatively rare in such conditions. Rolleston [2] states that "in typhoid fever the bacilli are always present in the gallbladder, but cholecystitis is comparatively infrequent." Osler [3] states that cholecystitis occurred in only nineteen of his series of 1,500 cases. (3) Theoretically, the mere presence of bacteria within the lumen of the gallbladder should be of no more significance in producing cholecystitis than that the presence of bacteria in the urine should imply an inflammation of the urinary bladder. It is well known that bacteria are frequently passed in the urine in large amounts without the coincidence of cystitis, especially in typhoid fever.

If the assumption is correct that the wall of the gallbladder beneath the mucosa is involved in most cases of cholecystitis, then it would seem reasonable to discard the idea of the serious importance of contact infections of the mucosa by organisms within the bile, particularly in view of the evidence outlined above. How then may an infection originate in the wall? It is obvious that in general only two routes are available for the transmission of organisms to the wall. One of these is the blood stream and the other is the lymph stream.

In 1909, Koch [4] demonstrated clumps of bacilli in the wall of a gallbladder and concluded that typhoidal cholecystitis is due to capillary embolism. In the same year, also, Chiarolanza,[5] working under Koch's direction, came to the same conclusion. Rosenow [6] has particularly emphasized the idea of the hematogenous origin of cholecystitis. Meyer and his collaborators,[1] in their exhaustive article on the mechanism of gallbladder infections, report experiments on 500 rabbits in which they attempted to produce a condition analogous to the human typhoid carrier state. They consider that the findings of Koch and Chiarolanza of bacterial emboli in the wall of the gallbladder during

2. Rolleston, H. D.: Diseases of the Liver, Gallbladder and Bile-Ducts, New York, the Macmillan Company, 1912, p. 608.

3. Osler, William: Practice of Medicine, 1905, p. 83.

4. Koch, J.: Typhusbazillen und Gallenblase, Ztschr. f. Hyg. u. Infectionskrankh, **62**:1, 1909.

5. Chiarolanza: Experimentelle Untersuchungen ueber die Beziehungen der Typhusbazillen zu der Gallenblase und der Gallenwegen, Ztschr. f. Hyg. u. Infectionskranskh, **62**:12, 1909.

6. Rosenow, E. C.: The Etiology of Cholecystitis and Gallstones and Their Production by the Intravenous Injection of Bacteria, J. Infec. Dis. **19**:527 (Oct.) 1916.

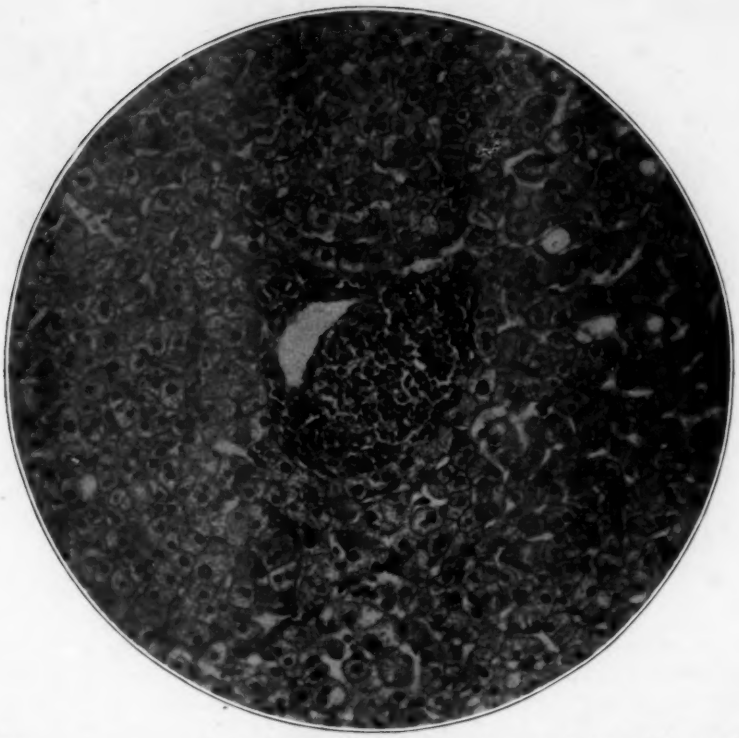

Fig. 1.—A common type of hepatitis occurring in association with a subacute cholecystitis. The periportal sheaths are densely infiltrated with leukocytes, chiefly of the polymorphonuclear variety. From a study of small pieces of liver removed at operation in cases of cholecystitis, either with or without stones, it has been noted that some degree of hepatitis is a constant accompaniment of cholecystitis.

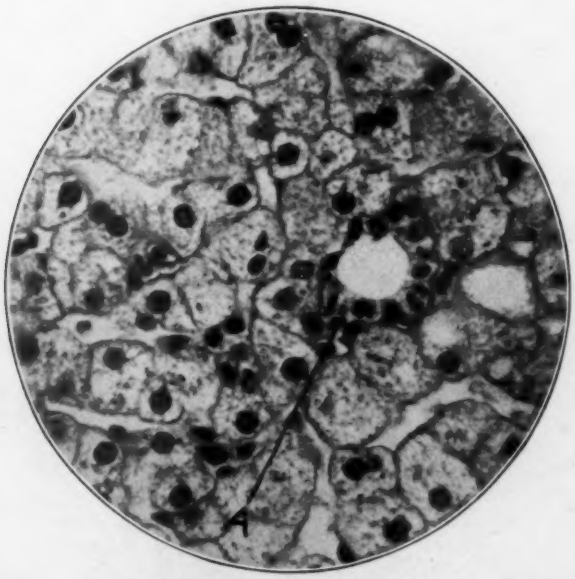

Fig. 2.—Higher power magnification of another part of the same liver showing accumulation of polymorphonuclear leukocytes around a small bile duct, indicated by line marked *A*.

Fig. 3.—Chronic hepatitis (biliary cirrhosis) in a woman who had a history of gallbladder trouble extending over a period of fifteen years. Jaundice had never been present; therefore, there had been no biliary obstruction. At operation, stones were found in the gallbladder. The marked thickening of the small bile ducts is strikingly evident.

Fig. 4. —Gallbladder removed at operation in case of cholecystitis without stones. Deep in the fibromuscular layers at a considerable distance from the mucosa were found collections of polymorphonuclear leukocytes, designated by lines marked *A*. These are usually much more conspicuous in the deeper layers than in the mucosa. Normally, the mucosa contains large numbers of round cells (lymphocytes).

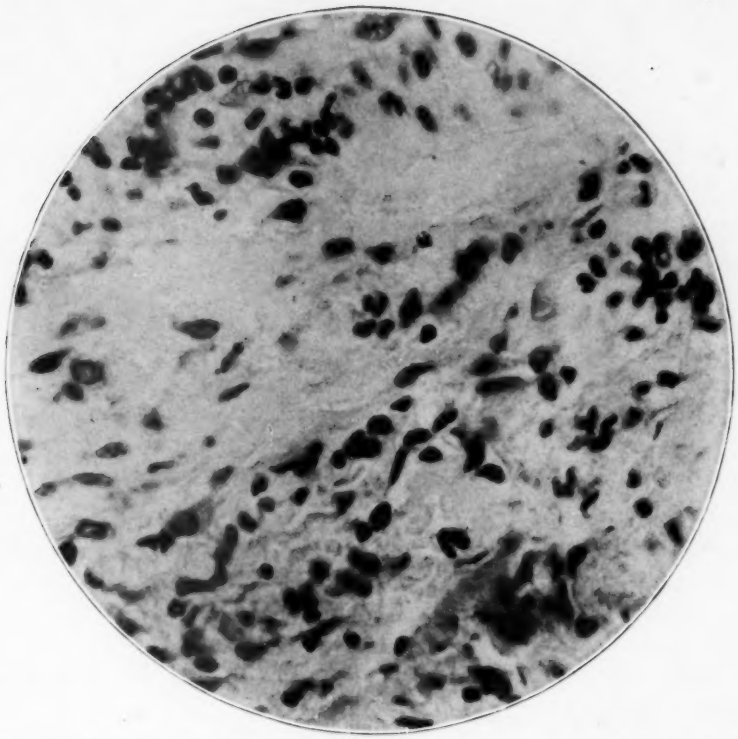

Fig. 5.—Higher magnification of same gallbladder as shown in Figure 4. The leukocytic infiltration shown here is deep in the wall of the gallbladder at a considerable distance from the mucosa.

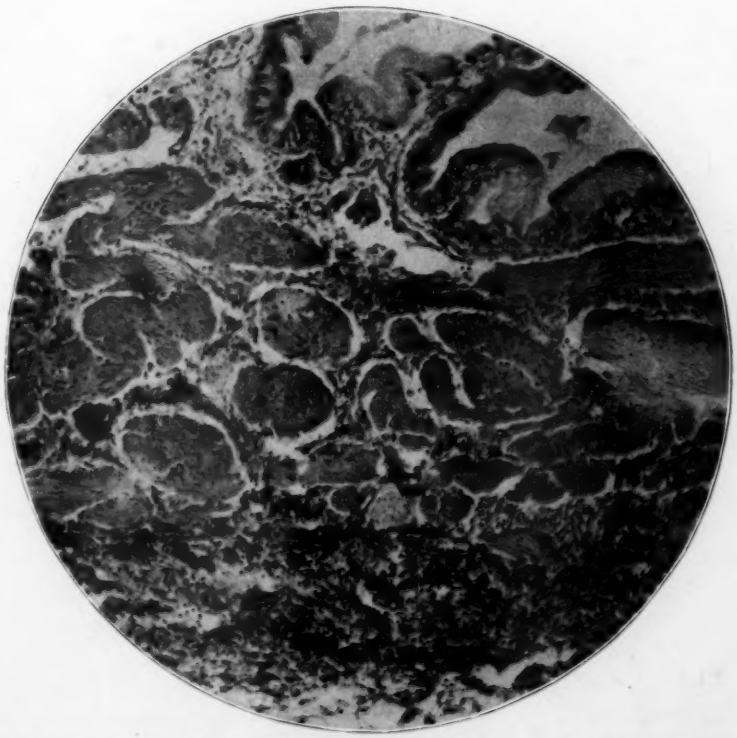

Fig. 6.—Another human gallbladder from case of cholecystitis without stones; wall extensively infiltrated with leukocytes; mucosa relatively intact.

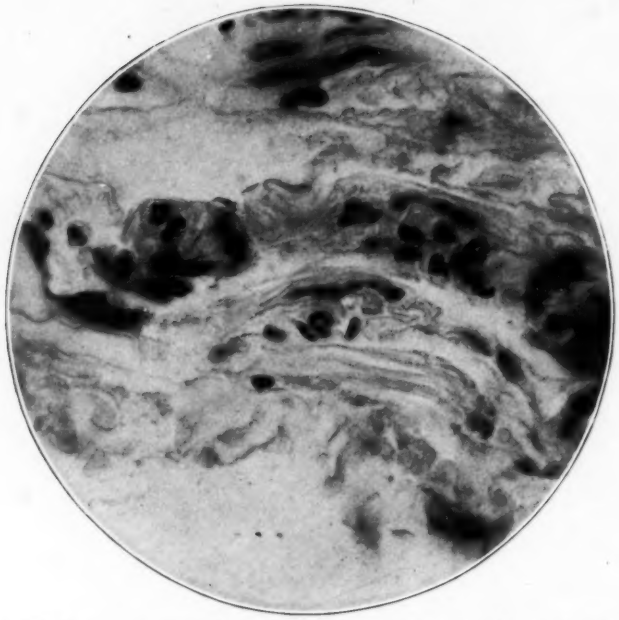

Fig. 7.—High power magnification of Figure 6 showing polymorphonuclear leukocytes far out in the wall of the gallbladder.

Fig. 8.—Another human gallbladder; cholecystitis without stones; mucosa relatively intact, but in outer layers of wall there is rather extensive leukocytic infiltration.

typhoid to be "exceptionally rare" and concerned with an "exceedingly severe and unique form of typhoid cholecystitis." They arrive at this conclusion partly because in their own experiments, even after the intravenous injection of enormous doses of typhoid bacilli (8,000 to 24,000 million), only about one third of the rabbits presented histologic evidence of an infection through the terminal capillaries of the mucosa, and also because, as they state, examination of human gallbladders in cases of death from typhoid shows a capillary embolism only occasionally. They are inclined, however, to consider very seriously, especially in the rabbit, the possibility of contact infection of the mucosa of the gallbladder by bacteria carried down in the bile from the liver, and they hold the opinion that deep infections of the wall are frequently the result of a spread of the infection from the mucosa. In human typhoid carriers, they consider that "the embolic infection of the gallbladder wall plays an insignificant rôle." They state, furthermore, that "a condition analogous to that found in man apparently exists in the guinea-pig. The microscopic and cultural study of a limited number of gallbladders derived from infected guinea-pigs convinces us that the wall is rarely, and then only slightly, infected through the bile." This then seems to constitute another argument against the frequency of contact infection of the gallbladder in man. There can be little doubt apparently that cholecystitis not infrequently is hematogenous in origin; but yet we do not believe that an explanation based on this idea is sufficient for most cases of spontaneous cholecystitis, especially those which are not typhoidal in origin. The well known, striking tendency of cholecystitis to be associated with appendicitis and other inflammatory lesions of the portal system must be something more than a coincidence. Birch-Hirchfeld,[7] in 1895, considered that organisms were carried to the liver and from there to the gallbladder by the bile. Since then, the "hematohepatogenous" idea of the origin of cholecystitis has been a popular one. But, as we have already shown, the idea of a contact infection of the mucosa of the gallbladder has very little to support it, at least so far as its being a frequent occurrence in man. Furthermore, it is difficult to explain the association of appendicitis and other portal infections with cholecystitis on the basis of a hematogenous infection of the wall of the gallbladder, because there is no direct path from the appendix to the gallbladder by way of the blood stream. The entire arterial supply of the gallbladder comes from the hepatic artery. The cystic artery supplies most of the blood; but there are in addition a few small branches of the hepatic artery which reach the gallbladder through its attachment to the liver. The veins of the gallbladder all empty into the portal vein. If organisms, therefore, are to be carried from the appendix to the gallbladder by the blood stream, they must

7. Birch-Hirchfeld: Quoted by Meyer et al, Footnote 1.

either travel through the whole systemic circulation back through the hepatic artery to the gallbladder or there must be a retrograde passage of them from the liver into the gallbladder through the tributaries of the portal vein. If the first alternative were correct, then there would be no more reason for cholecystitis to be associated with appendicitis than with infections elsewhere in the body, as the hand for example. The second alternative is based on the slender support of a comparatively rare possibility, a retrograde thrombosis, or at least a retrograde pylephlebitis.

There is, however, one route between the appendix and gallbladder which is comparatively direct and easy to understand, and that is a "hematolymphatic" route. Sudler [8] has shown a very intimate lymphatic connection between the liver and gallbladder through the attachment of the gallbladder to the liver. This is shown in Figure 34, which is taken from his article. We have repeated some of Sudler's experiments and have confirmed his idea of the general arrangement and anastomosis of the hepatic and cholecystic lymphatics. The lymph vessels of the gallbladder may be seen after injections of such a substance as Prussian blue into the portal vein. Conversely, also, injections made into the wall of the gallbladder not only follow the lymphatics into the liver in the interlobular sheaths but also may be seen to pass along the lymphatics of the common duct and into the pancreas. An infection of the liver, therefore, would tend to spread to the gallbladder by way of the lymphatics, and conversely an infection of the gallbladder would tend to spread not only to the liver but also along the common duct to the pancreas. In this respect, infections in this region would merely follow the rule of infections elsewhere in the body. We should expect to find evidence, therefore, that in cases of cholecystitis there is also an associated inflammation in the liver.

In 1918, one of us [9] showed in a series of cases in which small pieces of the liver were removed at operation for cholecystitis that microscopic evidence of hepatitis existed in every case. The inflammation was chiefly in the interlobular sheaths and appeared to be a pericholangitis. This location of inflammatory changes is in harmony with the idea of a lymphangitis, for it is here that the hepatic lymph vessels are chiefly situated.[10] In another paper, we [11] showed that in the

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8. Sudler: The Architecture of the Gallbladder, Bull. Johns Hopkins Hosp. **12**:126, 1901.

9. Graham, E. A.: Hepatitis: A Constant Accompaniment of Cholecystitis, Surg., Gynec & Obst. **26**:521 (May) 1918.

10. Mall, F. P.: On the Origin of the Lymphatics in the Liver, Bull. Johns Hopkins Hosp. **12**:146, 1901.

11. Peterman, M. G.; Priest, W. S., Jr., and Graham, E. A.: The Association of Hepatitis with Experimental Cholecystitis and Its Bearing on the Pathogenesis of Cholecystitis in the Human, Arch. Surg. **2**:92 (Jan.) 1921.

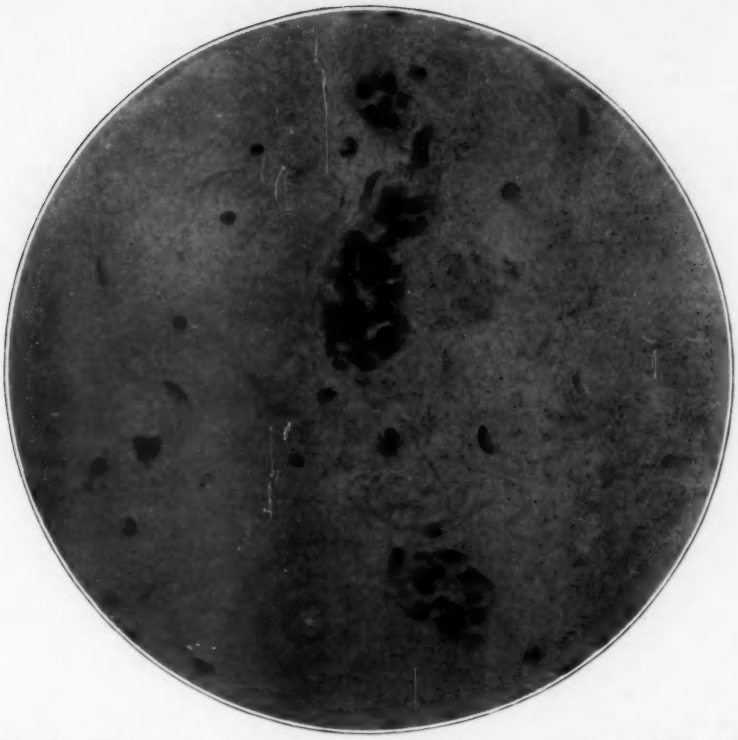

Fig. 9.—High power magnification of another human gallbladder showing small accumulations of polymorphonuclear leukocytes far out in the wall.

Fig. 10.—Cholecystitis with stones; whole wall, including mucosa, markedly inflamed.

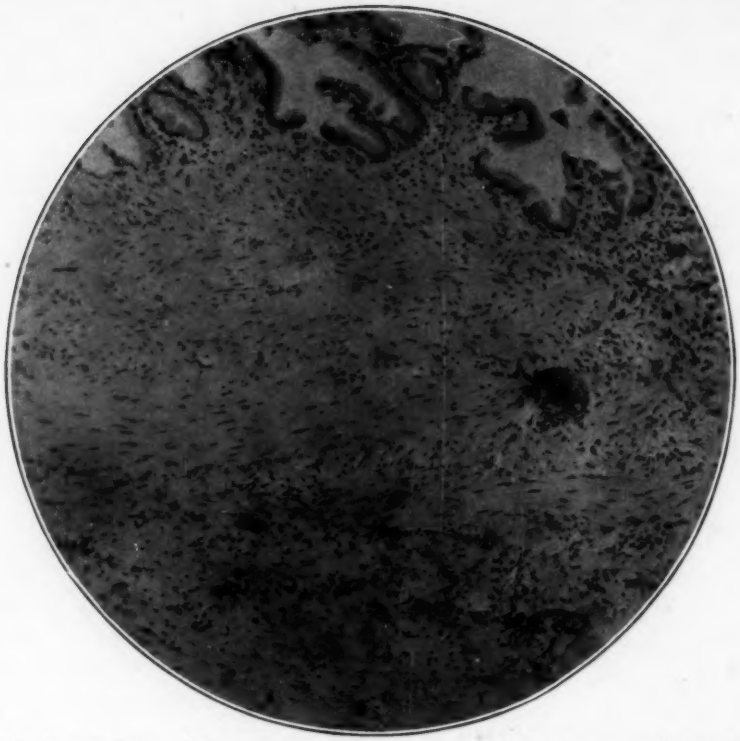

Fig. 11.—Cholecystitis without stones; mucosa relatively normal, but leukocytic infiltration in outer layers of wall.

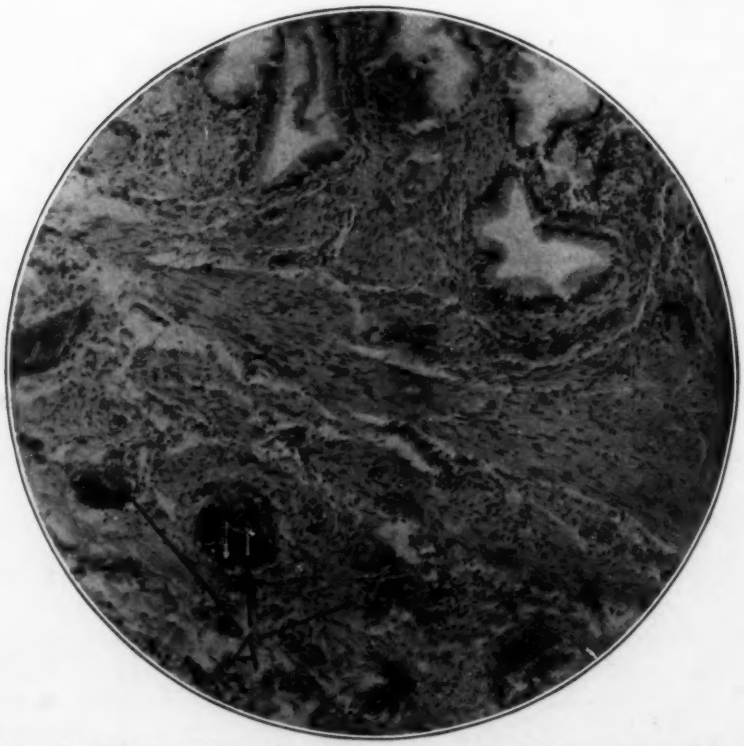

Fig. 12.—Cholecystitis without stones; normal mucosa but rather large collections of leukocytes (both polymorphonuclears and round cells) in deeper layers of wall, designated by lines marked *A*.

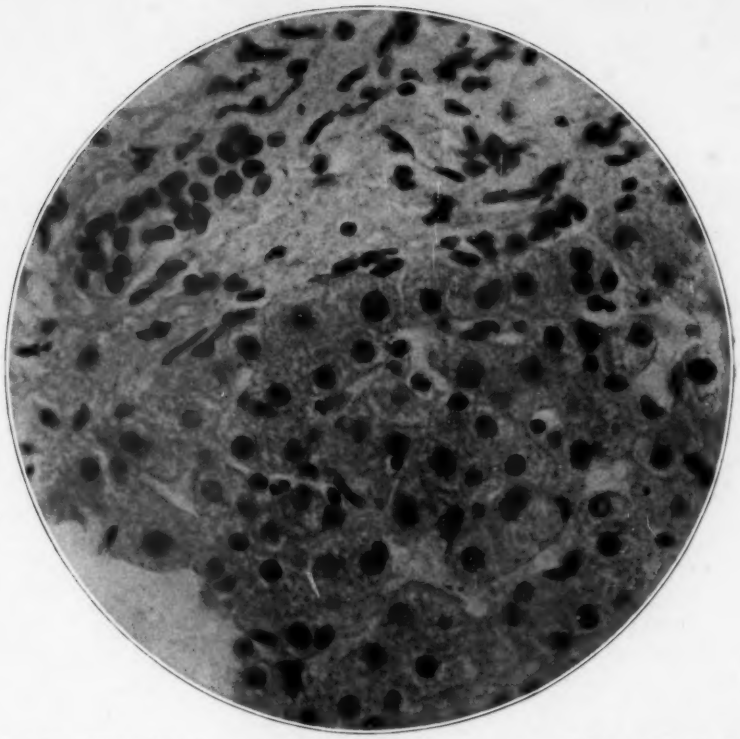

Fig. 13.—Hepatitis in association with chronic appendicitis; piece of liver removed at operation for chronic appendicitis; no evidence of cholecystitis. Note polymorphonuclears in lobule near its periphery.

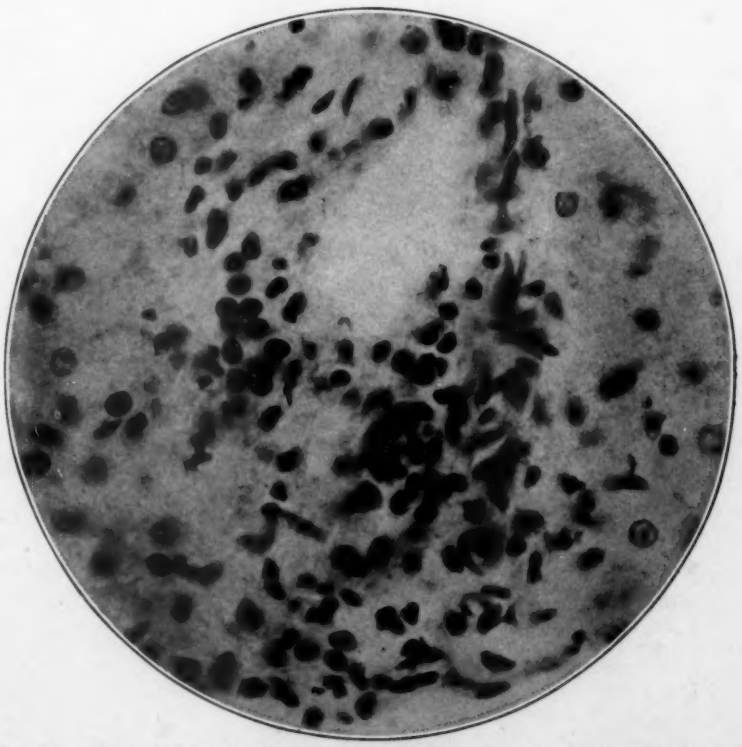

Fig. 14.—Hepatitis with chronic appendicitis, showing leukocytic infiltration in interlobular tissue; piece of liver removed in another case of chronic appendicitis; no evidence of cholecystitis.

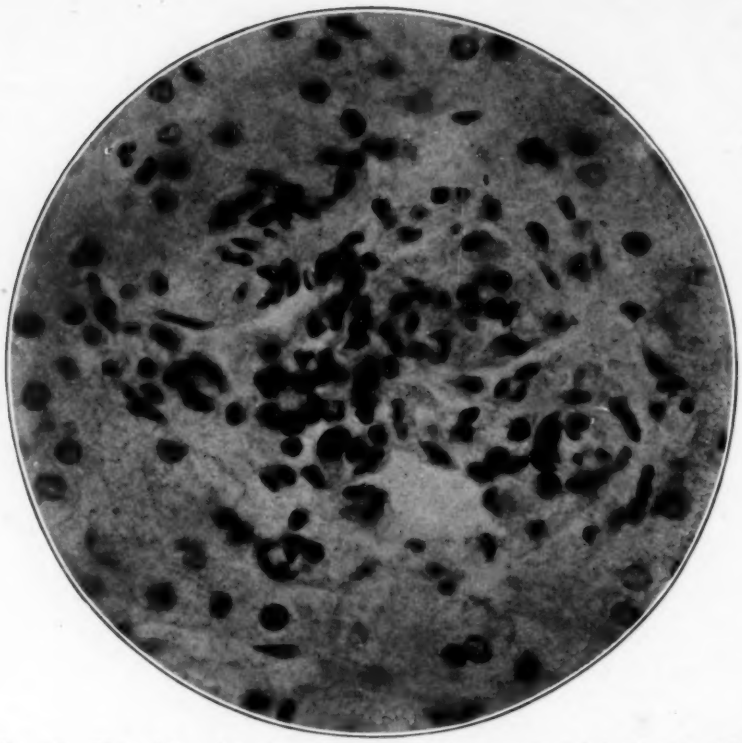

Fig. 15.—Hepatitis with chronic appendicitis; periportal leukocytic infiltration; piece of liver removed at operation for chronic appendicitis; no evidence of cholecystitis.

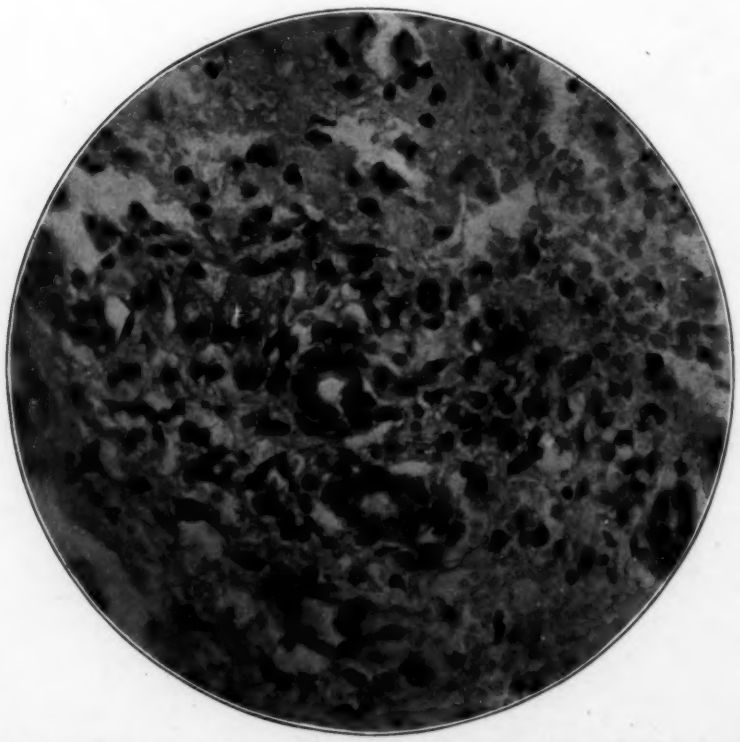

Fig. 16.—Experimental hepatitis by injection of portal vein with *Staphylococcus aureus* (Dog 1); marked periportal infiltration with polymorphonuclear leukocytes.

dog experimental cholecystitis is constantly accompanied by a hepatitis of the same sort as already observed in man. It is interesting that when this occurs secondarily to the production of a cholecystitis the right lobe of the liver is very much more affected than the left lobe, and the most marked changes of all are in the right lobe near the gallbladder. This all seems to indicate that the spread of the infection to the liver from an already inflamed gallbladder must occur through the lymphatics rather than through the veins, because in the latter instance, since the veins are tributaries of the portal vein, a more even distribution of the infection through the liver would be expected. The possibility of the establishment of a vicious circle between an infected gallbladder and the liver, whereby each may reinfect the other through the lymphatics, becomes readily apparent. This finding of the constancy of a hepatitis in association with cholecystitis has been confirmed recently by Judd,[12] who states, "A review of our studies inclines us to believe with Graham that cholecystitis rarely exists without hepatitis." In our original paper, to which reference has already been made, it was said that in a series of thirty consecutive cases coming to operation for gallbladder disease an enlargement of the liver was noted in 87 per cent. Subsequent observations have not borne out this high percentage, and we are forced to the conclusion that the actual percentage of noticeable enlargement of the liver is very much smaller than the figure given.

If we are to explain the frequent association of appendicitis with cholecystitis on the basis of a primary infection of the liver from the appendix, by way of the portal vein, and of a cholecystitis resulting from that secondarily, by way of a lymphatic spread from the liver to the gallbladder, then obviously it would seem necessary to find evidence of hepatitis in association with appendicitis. Recently, in a few cases which came to operation for chronic appendicitis, we removed small pieces of the liver for examination. Three of the accompanying photomicrographs (Figs. 13, 14 and 15) show areas of infiltration in the liver (both polymorphonuclears and round cells). In none of the cases represented here was there any evidence, either clinical or at operation, of cholecystitis. This fact seems important since in consequence the existing hepatitis could hardly be assumed to have been secondary to a cholecystitis. We have frequently noted slight liver tenderness in association with chronic appendicitis and have considered the sign of some importance in diagnosis. The question may arise as to why cholecystitis did not occur in the cases of hepatitis with appendicitis illustrated here. The answer is, of course, difficult, but perhaps the intensity of the hepatitis was not sufficient.

12. Judd, E. S.: Relation of the Liver and Pancreas to Infection of the Gallbladder, J. A. M. A. **77**:197 (July 16) 1921.

It is interesting that even those who favor the idea of the contact infection of the mucosa of the gallbladder by bacteria carried in the bile from the liver, the so-called "hematohepatogenous" theory, are forced to assume some degree of inflammation of the liver preceding the infection of the gallbladder. This question is discussed at some length by Meyer and his collaborators who state that "dosage, virulence and *lesions* [italics ours], and not the secretory, detoxifying activity of the liver, are the prerequisites for the passage of bacteria from the blood to the bile capillaries. . . . The conception of a purely mechanical passage deserves little consideration." Wyssokowitch [13] and also Blachstein [14] considered liver necroses prerequisites for the infection of the biliary passages in typhoid. If it is granted then that some degree of inflammation of the liver must be present before the bile can be infected, it seems only reasonable to assume that in many cases the infection will spread by means of the lymphatics and will therefore involve the gallbladder wall. Meyer states that "from an experimental standpoint the lymphatic route is of no importance," but yet he says that "in the course of peritonitis or other abdominal infections (appendicitis), streptococci can be transported through the lymphatics to the biliary passages," and again, "the so-called ascending route of infection is in all probability due to an invasion of bacteria through the lymphatics." He also states that in about 1 per cent. of the rabbits and guinea-pigs used there was a spontaneous cholecystitis due to *Bacillus coli*, streptococci or staphylococci. In these cases, "occasionally the bile appeared to be normal while the histologic picture exhibited a low-grade infection of the lymphatics of the mucosa and subserosa."

We believe that there is quite a little confusion about what is meant by the biliary lymphatics. Meyer and his collaborators speak of them as if they were entirely extrahepatic, and apparently they do not recognize that the extrahepatic lymphatics are continuous with those within the liver. This is the common conception of the lymphatics of the biliary tract. Sudler's important work is too little known. As a matter of fact, we do not see why nearly all the results on experimental cholecystitis cannot be just as easily interpreted on the basis of a lymphatic infection of the wall as on the idea either that there has been a contact infection of the mucosa or an infection of the wall directly through the blood stream. We admit, however, the possibility of infection through both of the latter means; but we are inclined to believe that the idea of a lymphatic origin secondary to a hepatitis explains the majority of cases of cholecystitis in man better than any other.

We have stated elsewhere that if a suitable substance, such as Prussian blue, be injected into the wall of the gallbladder, its course

13. Wyssokowitch: Quoted by Meyer et al, Footnote 1.
14. Blachstein: Quoted by Meyer et al., Footnote 1.

Fig. 17.—Experimental cholecystitis from Dog 1 by injection of portal vein; extensive inflammation of wall; mucosa relatively unaffected.

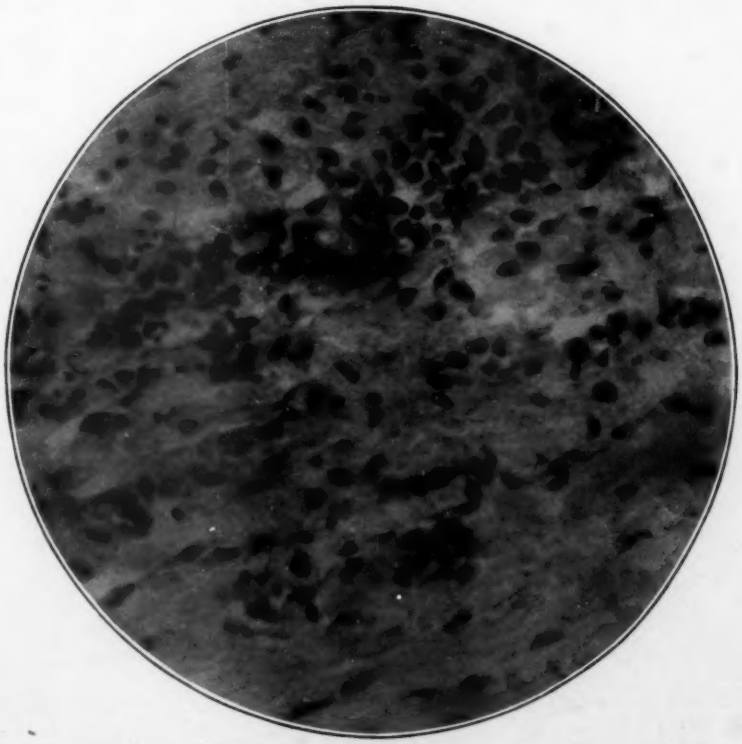

Fig. 18.—Higher power photomicrograph of specimen shown in Figure 17, showing leukocytic infiltration of wall of gallbladder.

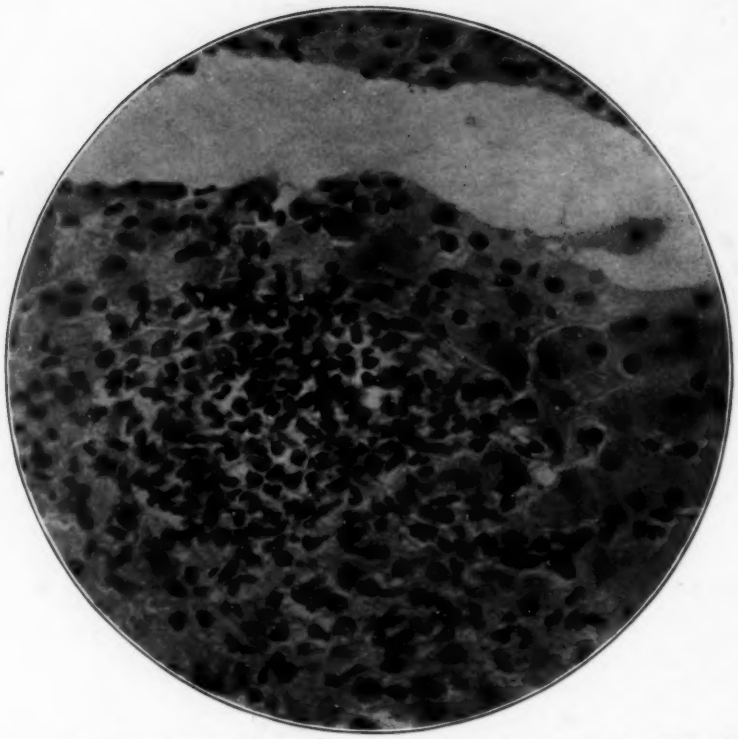

Fig. 19.—Experimental hepatitis by injection of portal vein (Dog 3).

Fig. 20.—Another section from same liver as shown in Figure 19.

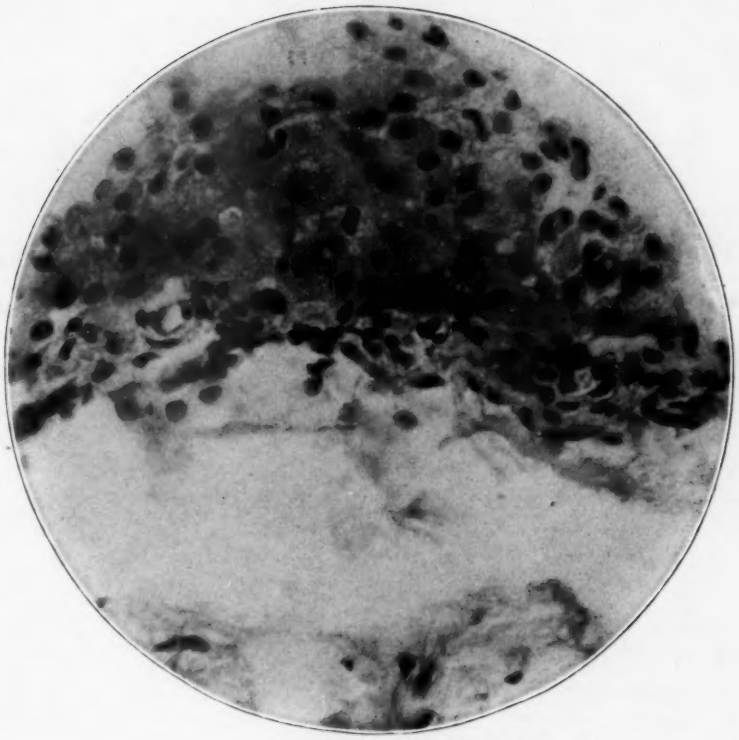

Fig. 21.—Experimental hepatitis (Dog 6).

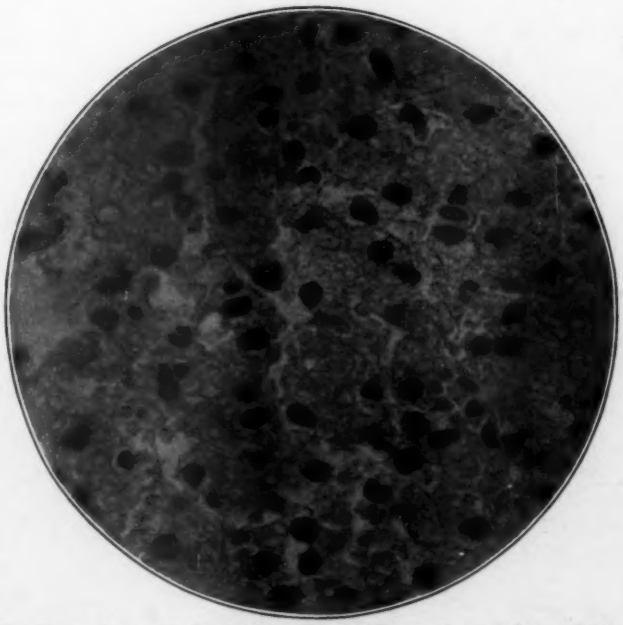

Fig. 22.—Experimental hepatitis (Dog 7); polymorphonuclear leukocytes scattered throughout whole lobule.

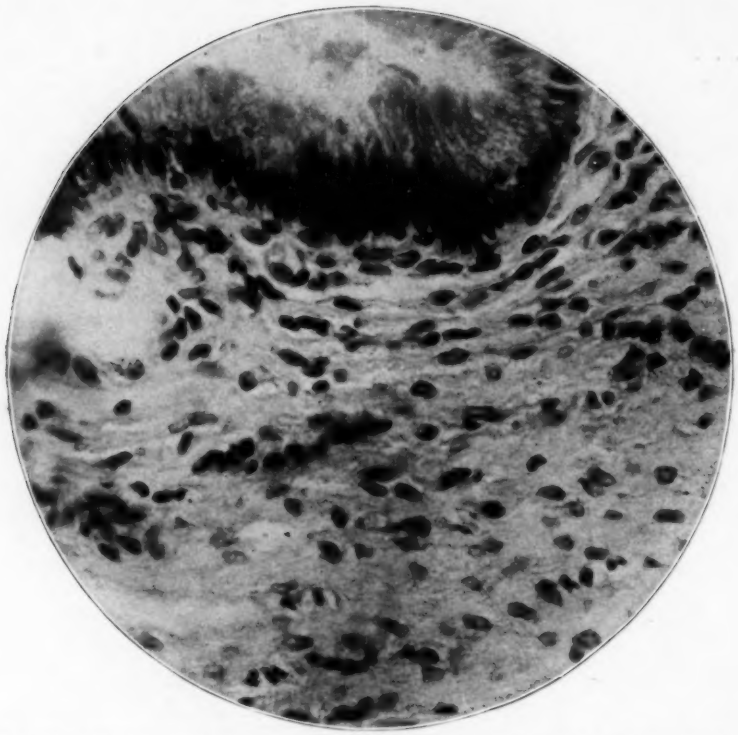

Fig. 23.—Cholecystitis in same dog (Dog 7); polymorphonuclear leukocytes scattered throughout wall of gallbladder.

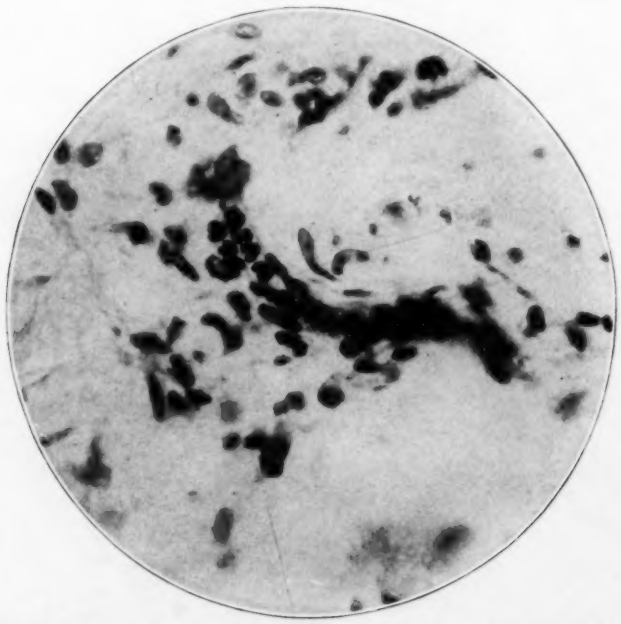

Fig. 24.—Same experiment as in Figure 23; polymorphonuclear leukocytes in deeper layers of wall of gallbladder.

may be followed through the lymphatics down along the common duct and into the head of the pancreas. For years, Deaver [15] has strongly advocated the lymphatic origin of the chronic interstitial pancreatitis which is so common an accompaniment of cholecystitis. Maugeret,[16] in 1908, wrote a thesis on this subject which offers much evidence in support of the possibility of the lymphatic spread to the pancreas. Archibald [17] has raised questions with regard to the lymphatic route theory and has sought to explain the pancreatitis on a basis of an influx of bile into the pancreatic ducts in a manner similar to Opie's [18] classical explanation of the origin of acute hemorrhagic pancreatitis. Deaver and Sweet [15] have replied to these objections of Archibald's in their recent article.

In animal experiments, we have obtained results which we believe tend to support the idea of the lymphatic origin not only of cholecystitis but also of inflammation of the common duct and of the pancreas. These experiments have consisted of several types and will be reported in full by one of us (Peterman), in an article soon to appear. Two types are of particular interest here because of their special bearing on the idea of the lymphatic origin of cholecystitis and its complications. One of these groups of experiments has already been reported [11] and referred to above. It consisted in the production of hepatitis secondary to experimental cholecystitis, induced by direct inoculation of rather large amounts of organisms into the lumen of the gallbladder after ligation of the cystic duct and artery. The hepatitis produced conformed to that which had already been seen in cases of cholecystitis in man, and, for the reasons given above, it was considered as most probably representing a lymphatic spread to the liver. The other type of experiment consisted in the production of hepatitis, cholecystitis, choledochitis and pancreatitis by portal vein injections. It was felt that if cholecystitis and its common complications could be interpreted on the basis of a lymphatic spread from a liver in which the periportal spaces were the seats of inflammation, then it should be possible often in a single experiment to produce by injection of the portal vein a simultaneous hepatitis, cholecystitis, choledochitis and pancreatitis. Not only has it been possible to do this repeatedly; but the types of lesion found have agreed with those which occur in spontaneous cases in

15. Deaver, J. B., and Sweet, J. E.: Prepancreatic and Peripancreatic Disease: With a Consideration of the Anatomic Basis of Infection from the Gallbladder to the Pancreas, J. A. M. A. **77**:194 (July 16) 1921.

16. Maugeret, R.: Cholécyste—Pancréatite, Thesis, Paris, G. Steinheil, 1908.

17. Archibald, E.: The Experimental Production of Pancreatitis in Animals as the Result of the Resistance of the Common Duct Sphincter, Surg., Gynec. & Obst. **28**:529 (June) 1919.

18. Opie, E. L.: Diseases of the Pancreas, Philadelphia, J. B. Lippincott & Co., 1903.

man. It has been possible also after the injections to demonstrate the infecting organisms in the liver, in the wall of the gallbladder, in the wall of the common duct and in the pancreas. The same results occurred regardless of whether the injections were made into the main trunk of the portal vein or into one of its radicles, as the appendix vein. We believe, therefore, that the results obtained are comparable to the cases of cholecystitis in man which may be considered as following appendicitis or some other lesion of the portal system.

The experiments were conducted on seven normal dogs. After anesthetization and preparation of the skin, a median laparotomy incision was made. The portal vein was exposed at its origin, and from 4 to 6 c. c. (depending on the size of the animal) of *Staphylococcus aureus* (twenty-four hour broth culture) was injected into the portal vein, and the abdomen was closed. These animals were observed daily, and four of them were killed and examined at necropsy, from two to six days afterward. Three of the dogs were given an additional injection of hemolytic streptococci because apparently they had not been much affected by the first injection of staphylococci. From two to five days after the second injection, these dogs were killed and necropsies performed. In two additional experiments, injections of streptococci were made into a radicle of the portal vein corresponding to the appendix vein in man, and similar results were obtained to those following the injections into the portal vein itself.

In a second set of experiments in which six rabbits were used, sections were stained and examined for bacteria after injecting streptococci into the portal vein. The cystic duct, including the cystic artery, was ligated in order to eliminate the possibility of the passage of bacteria down from the liver in the bile and also to reduce to a minimum the possibility of the injected organisms reaching the gallbladder wall by way of the blood stream. After ligation of the cystic artery, the only way by which the organisms could reach the gallbladder by the blood would be either through the few small branches of the hepatic artery which pass through the attachment of the liver to the gallbladder or by a retrograde thrombosis of the portal vein into the cystic veins (which are its tributaries). In order to minimize the latter possibility, the animals were killed and examined at intervals of one, two, twelve, twenty-four and forty-eight hours, respectively, after being injected. Except perhaps in the last instance, it was felt that the time intervals were too short to permit a retrograde thrombosis. Streptococci were found in all of the last four rabbits, not only in the liver and the wall of the gallbladder but also in the pancreas. The common duct was not searched. Results of an identical sort were obtained in three other rabbits in which the injection was made into a vein corresponding to the appendix vein in man. The only exceptions,

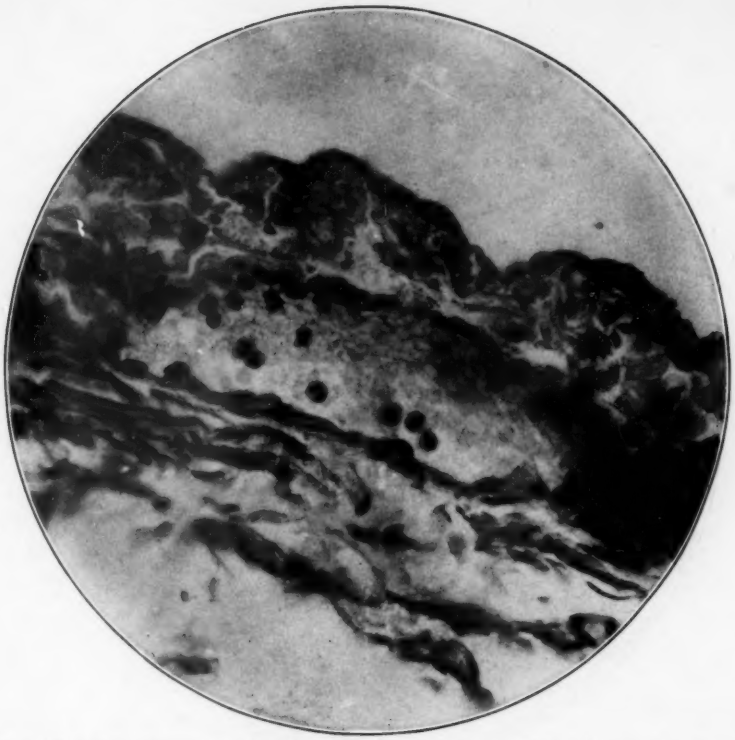

Fig. 25.—Experimental cholecystitis (Dog 7). This shows apparently a lymphatic vessel in the outer layer of the gallbladder containing polymorphonuclear leukocytes and presenting other evidence of acute inflammation.

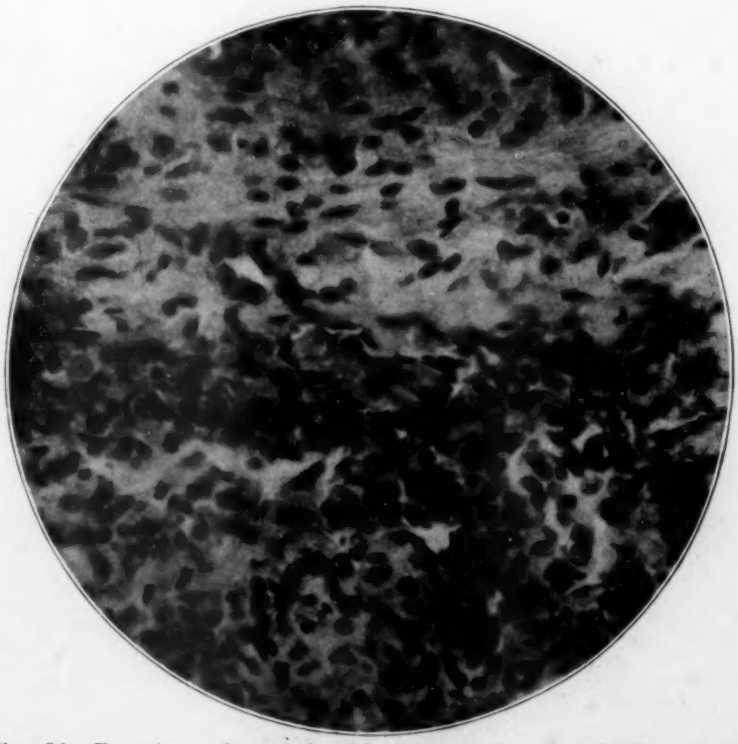

Fig. 26.—Experimental choledochitis by portal vein injection; extensive leukocytic infiltration of wall (Dog 3).

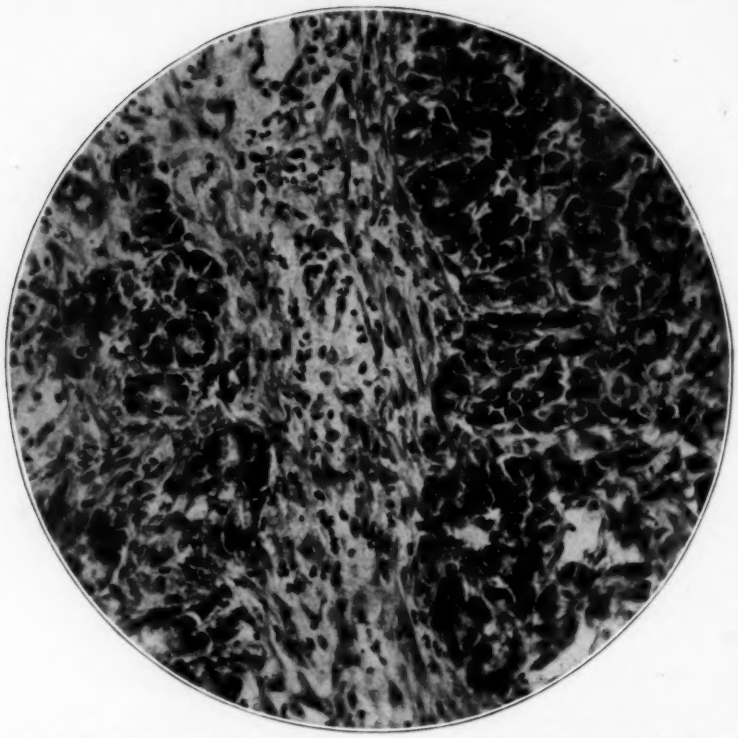

Fig. 27.—Experimental interstitial pancreatitis by portal vein injection (Dog 1); many leukocytes, chiefly polymorphonuclear, in trabeculae of pancreas.

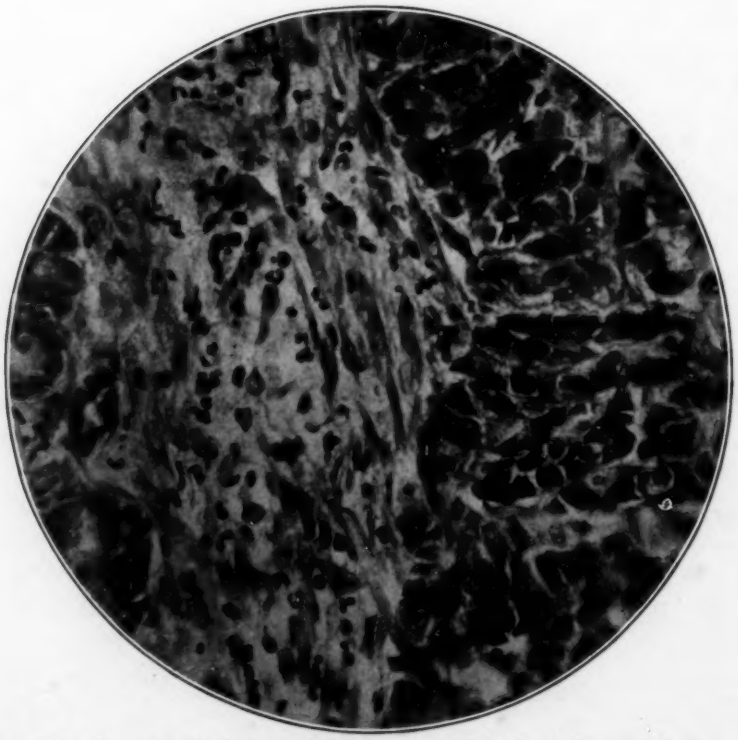

Fig. 28.—Experimental interstitial pancreatitis (Dog 2); marked preponderance of polymorphonuclear leukocytes over other cells in trabeculae.

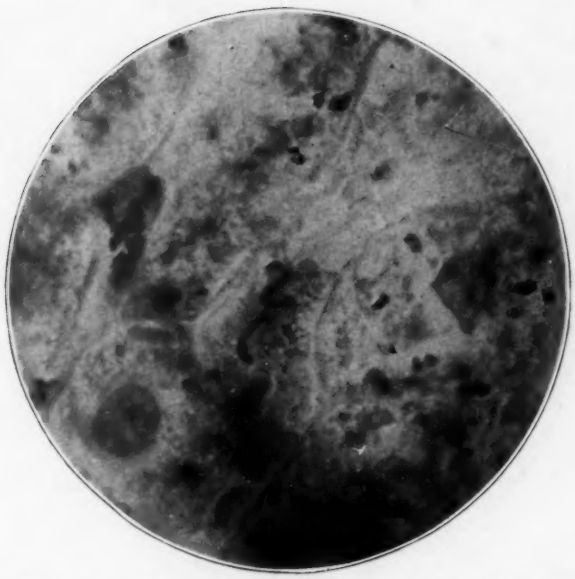

Fig. 29.—Experimental hepatitis in rabbit; portal vein injection with streptococci; demonstration, two hours later, of organisms in liver near attachment to gallbladder.

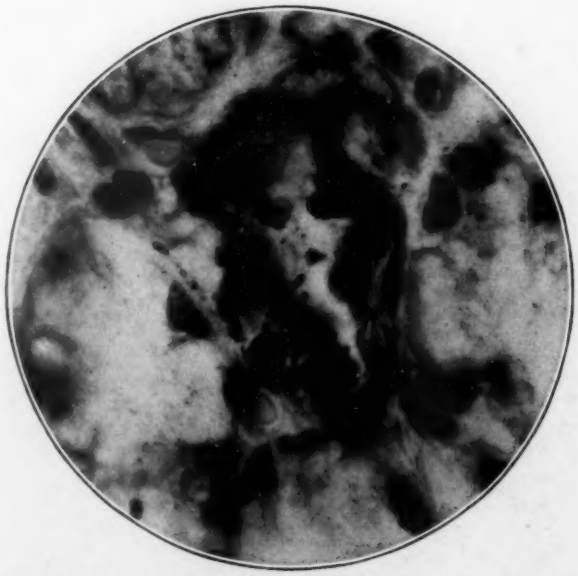

Fig. 30.—Experimental hepatitis in another rabbit after portal vein injection with streptococci; twelve hours later organisms found in liver near its attachment to the gallbladder.

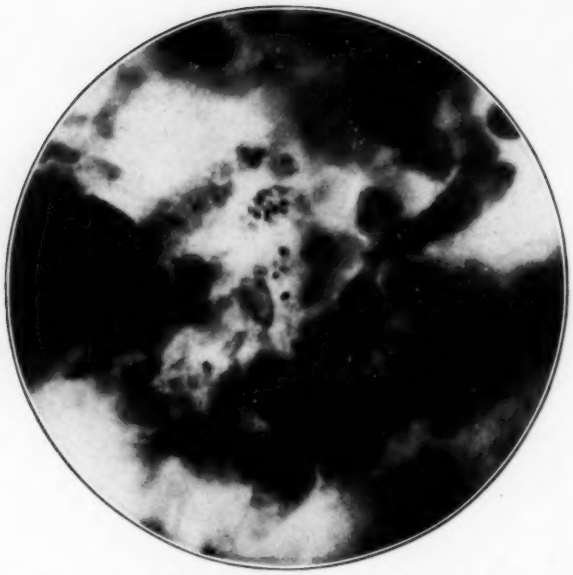

Fig. 31.—Experimental cholecystitis in rabbit; injection of portal vein after ligation of cystic duct and cystic vessels; two hours later organisms found in wall of gallbladder.

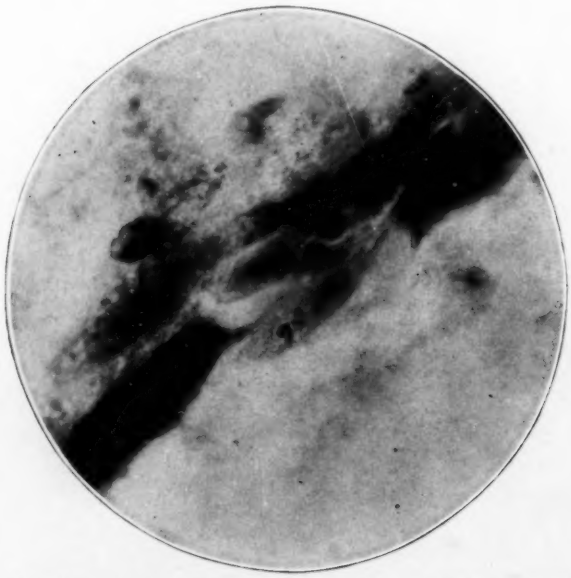

Fig. 32.—Experimental cholecystitis in another rabbit; organisms demonstrated in wall twelve hours after portal vein injection.

therefore, were in the rabbits which were killed one and two hours, respectively, after the injections. It seems to us that these findings harmonize well with what would be expected on the assumption that the organisms reached the gallbladder wall from the liver through the lymphatics in the attachment of the gallbladder to the liver, because apparently there was no other route of access available. Even if they came through the very doubtful route of the few small branches of the hepatic artery which pass through the attachment of the gallbladder to the liver, one would have to assume that they had passed through the circuit of the systemic circulation after leaving the liver and had come back through the hepatic artery. Each rabbit was injected with 2 c.c. of a twenty-four hour broth culture of hemolytic streptococci.

PROTOCOLS

EXPERIMENT 1.—Five c. c. of a twenty-four hour broth culture of *Staphylococcus aureus* was injected into the portal vein of Dog 1. The dog was killed six days after the injection.

Necropsy Findings.—Liver: It was somewhat swollen, a grayish-blue, and the edge was rounded. Microscopic: There was extensive diffuse hepatitis, but the most marked infiltration of polymorphonuclears was in the interlobular sheaths.

Gallbladder: It was bound to the liver by adhesions. Microscopic: There was marked inflammation, with polymorphonuclears scattered throughout the wall. The mucosa was also involved, but the epithelium was practically intact.

Common Duct: Acute inflammation was marked. The entire wall was infiltrated with polymorphonuclears and marked vascular dilatation.

Pancreas: There was rather extensive infiltration of the trabeculae of connective tissue with polymorphonuclear leukocytes and a few red cells. There was no parenchymatous necrosis, but acute interstitial pancreatitis existed.

EXPERIMENT 2.—Four c. c. of *Staphylococcus aureus* and four days later 6 c. c. of *Staphylococcus aureus* was injected into Dog 2. The dog was killed three days after second injection.

Necropsy Findings.—Liver: It was dark and edematous. Microscopic: There were marked fatty changes, especially in the center of the lobules. There was moderate infiltration of the periportal tissues with leukocytes, mostly polymorphonuclears, extending up into parenchyma.

Gallbladder: It was pale and adherent to the liver. Microscopic: There was extensive acute inflammation, with marked infiltration of the wall with polymorphonuclears, most pronounced in the outer layers, and marked vascular dilatation. The epithelium was practically normal. Staphylococcus was demonstrated in the walls.

Pancreas: There was marked polymorphonuclear infiltration of the connective trabeculae, and acute interstitial pancreatitis.

EXPERIMENT 3.—Five c. c. of *Staphylococcus aureus* and three days later a second injection of 2 c. c. of hemolytic streptococci were injected in Dog 3, which was killed five days after the second injection.

Necropsy Findings.—Liver: There were several scattered abscesses. Microscopic: There were moderate fatty changes, and very extensive infiltration of polymorphonuclear leukocytes throughout the whole liver but evidently begin-

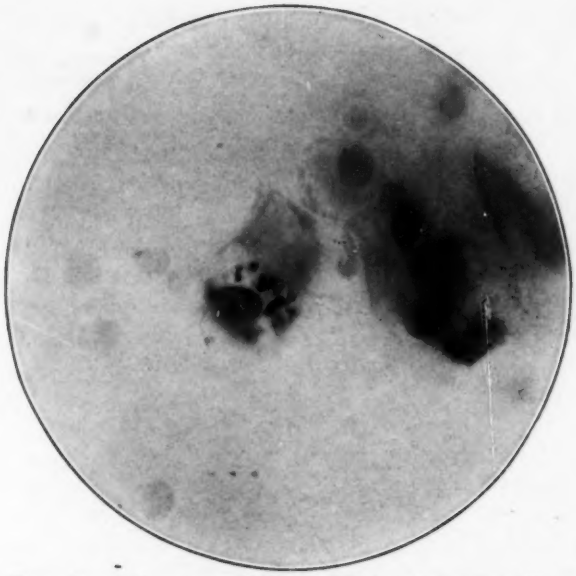

Fig. 33.—Experimental pancreatitis in rabbit; demonstration of organisms in pancreas twelve hours after injection of portal vein.

Fig. 34.—Anastomosis between lymphatics of gallbladder and liver (taken from Sudler). The extensive communication between the lymphatics of the liver and gallbladder is seen particularly at the sides of the gallbladder.

ning at the periphery of each lobule. In places there were marked accumulations of polymorphonuclear leukocytes and some mononuclear cells around the interlobular veins. There was very marked infiltration of polymorphonuclear cells beneath the liver capsule.

Gallbladder: It was adherent to the liver. The wall was gray. Microscopic: There was moderate infiltration of the wall with polymorphonuclear leukocytes and round cells, especially at the site of attachment between the liver and gallbladder.

Common Duct: The wall was extensively infiltrated with leukocytes. The mucosa was ulcerated.

Pancreas: There was polymorphonuclear infiltration into the trabeculae (moderate).

EXPERIMENT 4.—Five c. c. of *Staphylococcus aureus* and three days later a second injection of 2 c. c. of hemolytic streptococcus were injected into Dog 4, which was killed two days after second injection.

Necropsy Findings.—Liver: There was no gross change. Microscopic: There was diffuse hepatitis but again it was more marked at the peripheries of the lobules and in the interlobular tissues.

Gallbladder: It was adherent to the liver. The wall was grayish-pink. Microscopic: There was rather marked leukocytic infiltration of the wall, especially in the outer layers and more particularly in the aspect of the gallbladder away from the liver (that part covered by peritoneum). This is where most of the lymphatics are (Poirier and Charpy). Mucosa was not particularly affected.

Pancreas: The same type of lesion as already described under earlier experiments was found here.

Dogs 5, 6, and 7 showed lesions essentially similar to those already described. Dog 5 received 3 c. c. of hemolytic streptococcus three days after an initial injection of 5 c.c. of staphylococcus. In Dogs 6 and 7 there were fibrinous adhesions between the gallbladder and the liver.

SUMMARY

In many cases, probably in a majority, cholecystitis represents a direct extension to the wall of the gallbladder from a liver already inflamed. The hepatitis usually begins and is most marked in the interlobular, or periportal, tissues; and it is apparently due to infection brought to the liver by the portal vein, and, more rarely perhaps, by the hepatic artery. A pericholangitis then occurs, and because of the intimate anastomosis between the lymphatics of the intrahepatic and extrahepatic biliary systems a direct extension into the wall of the gallbladder takes place as well as into the common duct and the pancreas. From the antecedent hepatitis, therefore, a cholecystitis, choledochitis and pancreatitis can be understood to occur if in the consideration of inflammations in this locality we apply the well known fact concerning inflammation in general, namely, that it extends by way of the lymphatics. We believe that the ideas expressed in this article explain more readily than any others heretofore offered the frequent and well recognized association of biliary tract infections with

lesions of the portal system (appendicitis, peptic ulcer, typhoid fever, suppurating hemorrhoids, etc.). It has been shown also that hepatitis may follow the experimental production of cholecystitis by direct inoculation of organisms into the gallbladder. This possibility suggests that, frequently perhaps, a vicious circle between the gallbladder and liver is established whereby each may reinfect the other; and it forcibly emphasizes the desirability of cholecystectomy rather than cholecystostomy because by this means the vicious circle can most readily be broken. The ideas here expressed also furnish added evidence of the necessity of treating, in addition to the gallbladder, any coexisting lesions of the portal system.

While the opinion is expressed here that many, and perhaps the majority of, cases of cholecystitis represent a lymphatic spread from the liver, we recognize also that some are doubtless hematogenous in origin, some perhaps are contact infections from bacteria carried down in the bile and a few perhaps may have originated in an ascending infection of the common duct through its lymphatics or have been due to a chance contact of a gallbladder with an inflamed contiguous organ.

NOTE.—Since writing this article we have seen an article by George P. Muller on the "Pathology of Cholecystitis" (Brooklyn M. J. **19**:11, 1905) in which he suggested in 1904 the lymphatic origin of cholecystitis. He states, "It is reasonable, therefore, to conclude that infection from the portal vein may reach the gallbladder through the superficial collecting lymphatics as well as from the bile." He apparently considers this rather a rare occurrence, however, as he states:

> Clinically, attention has been called by Ochsner and others to the association of gallstones and appendicitis, but in the rather extensive series of gallbladder operations performed by Dr. Deaver, we have found but one case in which the bile infection might be directly traceable to a diseased appendix, while upon the other hand in several thousand operations upon the appendix the connection is quite as limited.

CLASSIFICATION AND MECHANISM OF FRACTURES OF THE LEG BONES INVOLVING THE ANKLE

BASED ON A STUDY OF THREE HUNDRED CASES FROM THE EPISCOPAL HOSPITAL *

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ANATOMIC AND SURGICAL STUDY

By Dr. Ashhurst

Pott, Dupuytren, Cooper, Maisonneuve, Tillaux, Hönigschmied, Stimson, Destot, Chaput, Quénu—and shall I not add Scudder, Cotton, Roberts and Speed? What can any one say more, at this late day? And yet the fact remains that there is no entirely satisfactory classification of ankle fractures in existence, and that many points of the mechanism of their production still are in dispute. I say there is no entirely satisfactory classification of these fractures in existence, because that which is the best, being the most scientific and complete, namely that of Quénu, is less a classification than a catalog; and because his strict adherence to an anatomopathologic classification, and his stern rejection of the historical pathogenetic classification, compel him to place side by side lesions in no sense related, except that the fracture lines happen to be similar, and to separate widely other lesions which, though presenting very dissimilar lines of fracture, nevertheless represent only different degrees or stages, or merely variants, of one and the same lesion.

It is perhaps unnecessary to argue the desirability of classification; for without classification, the relation of one lesion to another can be neither remembered nor understood in any department of knowledge; and comprehension is a prerequisite for intelligent memory and for rational diagnosis and treatment.

HISTORICAL

Si chirurgien experimenté que vous soyez, ne sautez pas ces premières pages. Elles sont indispensables, mais fatigantes à lire; mieux vaut remettre à un autre jour cette besogne ardue que de l'entreprendre mal disposé.—Farabeuf.

Pott [1] (1769) described a fracture which doesn't exist, and Dupuytren [2] (1819) commended him for his acute observation and fidelity

* Read before the American Surgical Association, June 14, 1921.

1. Pott: Some Few General Remarks on Fractures and Dislocations, London, 1769, p. 57.

2. Dupuytren: Ann. méd.-chir. d. hôp. et hosp. civ. de Paris, 1819, p. 1.

to nature. Cooper [3] (1822), more sensible than either, merely recounted what he had seen, avoided speculation about what he had not seen, and was silent on subjects about which he had no knowledge. Maisonneuve [4] (1840) and Tillaux [5] (1872) studied the mechanism by experiments on the cadaver, and though they were both correct, they came to different conclusions; and Tillaux thought his work had entirely invalidated that of Maisonneuve. Hönigschmied's [6] (1877) experiments on cavaders (125 in all) may be said to have carried this means of investigation to its limit; and though subsequent students, including myself, have repeated such experiments, it is evident that this method of investigation has many limitations, as it is impossible to reproduce in the cadaver the falls which living patients suffer and the muscular tension to which their limbs are constantly subjected.

Stimson's [7] study, in 1892, was the last one of importance before the advent of the roentgen ray; and it was Destot [8] (1911) who first of all published extensive observations of ankle fractures illustrated by, and based on, roentgenographic studies. It is strange that no English writer since Cooper has made a special study of fractures at the ankle, and that with the exception of Stimson, already mentioned, no American surgeon has made a particular study of the subject, though Scudder, Cotton, Roberts and Kelly, and Speed have discussed it at greater or less length in their textbooks on fractures. It is evident, however, to any attentive student of the subject that most of the recent writers, if not, indeed, all, demonstrate by their statements that they have not themselves read, or at any rate have not read with understanding, the works of their predecessors—especially of Dupuytren, Cooper, Maisonneuve and Tillaux. Most surgeons think they know what they mean by a "Pott's fracture," but as I said before, there is little use in knowing much, or anything, about a type of fracture which does not exist.

What is Pott's fracture? Let Mr. Percivall Pott tell in his own words (1769).[9]

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3. Cooper: Treatise on Dislocations and Fractures of the Joints, London, 1822.

4. Maisonneuve: Arch. gén. de méd. **1**:165, 433, 1840.

5. Tillaux, cited by Gosselin: Bull. de l'Acad. de méd., Paris, Series 2 **1**: 817, 1872.

6. Hönigschmied: Deutsch. Ztschr. f. Chir. **8**:239, 1877.

7. Stimson: New York M. J. **55**:701, 1892.

8. Destot: Traumatismes du pied et rayons-x, Paris, 1911.

9. It should be borne in mind that Pott is discussing the importance of treating fractures of the leg bones in the flexed position (the limb resting on its outer surface with the knee bent), and hence that he speaks of fractures of the fibula only incidentally.

"The limb most commonly preserves its figure and length . . . if the fibula only be broken, in all that part of it which is superior to letter *A* in the annexed figure" (Fig. 1), "or in any part of it between its upper extremity, and within two or three inches of its lower one. . . . I have already said . . . that the support of the body and the due and proper use . . . of the joint of the ancle depend almost entirely on the perpendicular bearing of the tibia upon the astragalus, and on its firm connection with the fibula. If either of these be perverted or prevented, so that the former bone is forced from its just and perpendicular position on the astragalus, or if it be separated by violence from its connection with the latter, the joint of the ancle will suffer a partial dislo-

Fig. 1.—Illustration from Pott's work, "Some Few General Remarks on Fractures and Dislocations," London, 1769, facing p. 69.

cation internally; which partial dislocation cannot happen without not only a considerable extension or perhaps laceration of the bursal ligament of the joint, which is lax and weak, but a laceration of those strong tendinous ligaments which connect the lower end of the tibia with the astragalus and os calcis, and which constitute in great measure the ligamentous strength of the joint of the ancle.

"This is the case when, by leaping or jumping, the fibula breaks in the weak part already mentioned, that is, within 2 or 3 inches of its lower extremity. When this happens, the inferior fractured end of the fibula falls inward, toward

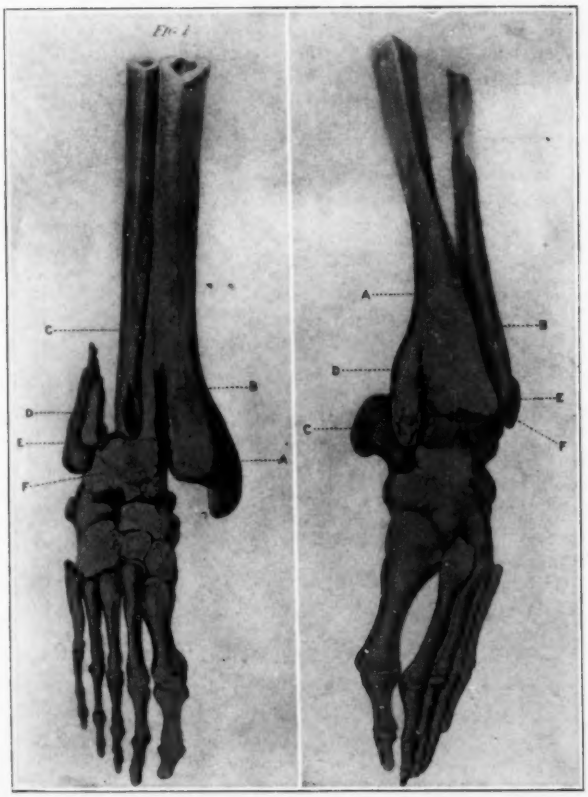

Fig. 2.—Illustrations from Cooper's "Treatise on Dislocations and Fractures of the Joints," London, 1822, Plate XVI, Figs. 1 and 2. "Fig. 1 shows the dislocation of the tibia inwards at the ankle-joint: *A*, the malleolus internus of the tibia thrown on the inner side of the astragalus; *B*, a portion of the tibia split off; *C*, fibula broken; *D*, the broken portion of the tibia adhering by ligament to the fibula; *E*, the malleolus externus of the fibula, with the broken portion of the tibia adhering to it, and *F*, astragalus thrown outwards. Fig. 2 shows the dislocation of the tibia outwards, at the ankle-joint: *A*, the tibia; *B*, the fibula; *C*, the os calcis; *D*, fracture of the tibia at the malleolus internus, which has become reunited; *E*, extremity of the fibula broken, and, *F*, tibia thrown on the outer side of the articulatory surface of the astragalus, to which it is anchylosed."

the tibia, that extremity of the bone which forms the outer ancle is turned somewhat outward and upward, and the tibia, having lost its proper support and not being of itself capable of steadily preserving its true perpendicular bearing, is forced off from the astragalus inward [Evidently, he means by the weight of the body, after primary fracture of the fibula produced by the first impact of the foot with the ground.] by which means weak bursal, or common

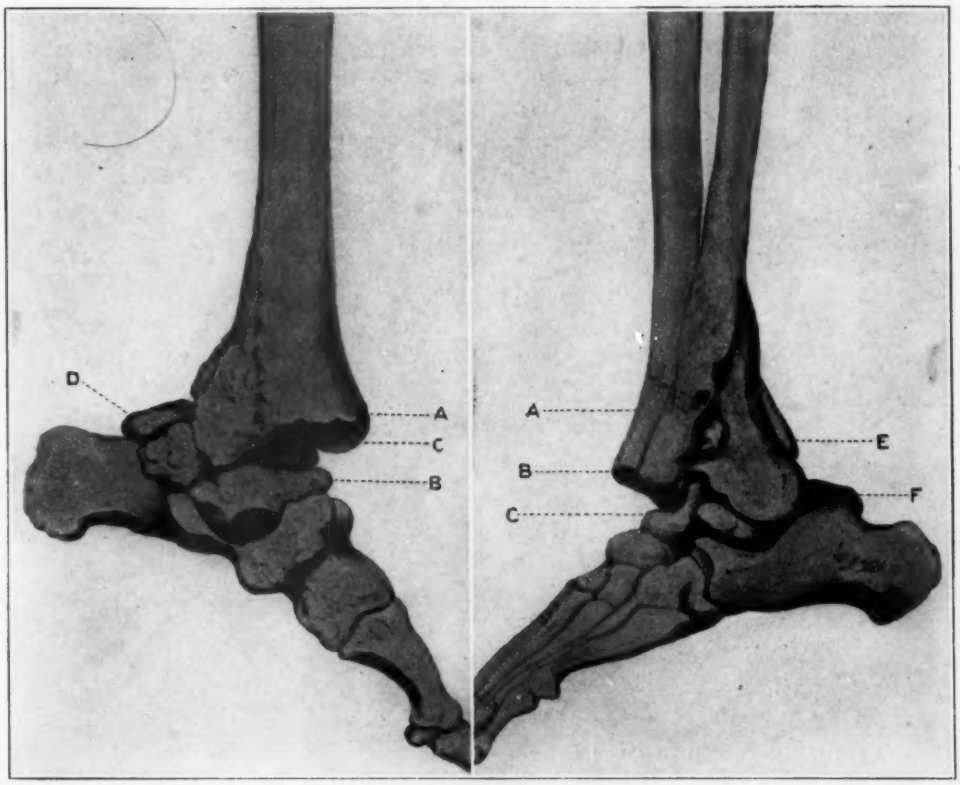

Fig. 3.—Cooper's Plate XVII, Figs. 1 and 2, "Partial dislocation of the tibia forwards, at the ankle-joint: Fig. 1: *A*, the tibia thrown forward over the os naviculare; *B*, the astragalus; *C*, new articulatory surface of the tibia, and, *D*, the portion of the astragalus behind the tibia. Fig. 2: opposite view of Fig. 1: *A*, the tibia thrown forwards; *B*, new articulatory surface of the tibia; *C*, astragalus; *D*, fibula broken and reunited; *E*, malleolus externus of the fibula, and *F*, astragalus behind the tibia."

ligament of the joint is violently stretched, if not torn, and the strong ones, which fasten the tibia to the astragalus and os calcis, are always lacerated, thus producing at the same time a perfect fracture and a partial dislocation, to which is sometimes added a wound in the integuments, made by the bone at the inner ancle. By this means, and indeed as a necessary consequence, all the tendons which pass behind or under, or are attached to the extremities of the tibia and fibula, or os calcis, have their natural direction and disposition so

altered, that instead of performing their appointed actions, they all contribute to the distortion of the foot, and that by turning it outward and upward." (Note that there is not a word of posterior displacement, and that this is not shown in the plate, here reproduced as Figure 1.)

This description, and the accompanying illustration, call for several remarks. It is to be noted, first, that Pott's fracture, as described and pictured by himself, is a primary, nearly transverse, fracture of the fibula, attended by a subsequently produced "partial dislocation" of the

Fig. 4.—Transverse section of leg through inferior tibiofibular joint, showing fibula lodged in its groove formed by the anterior and posterior tubercles of the tibia. Note the obliquity of the intermalleolar axis, forming an angle of 30 degrees with the axis of motion of the ankle joint, which is nearly transverse. Note that the anterior tubercle projects laterally much farther than the posterior, so that in anteroposterior roentgenograms its shadow much overlaps that of the fibula. (From a preparation in the laboratory of operative surgery, University of Pennsylvania.)

ankle joint internally. Second, the fracture of the fibula occurs at, or below, the point marked *A* in the illustration ("within 2 or 3 inches of its lower extremity"); and the upper end of the lower fragment is described as falling in against the tibia, while the external malleolus is turned outward. Third, there then is supposed to occur rupture of

the ligaments below the internal malleolus, with a partial dislocation of the tibia inward, off the trochlea of the astragalus, as the latter bone turns outward, around a more or less anteroposterior axis. Now, such a fracture of the fibula does not occur as a type, as may be readily verified by the examination of any series of roentgenograms or post-mortem specimens; and if it did occur, it would be impossible for the upper end of the lower fragment to fall in against the tibia, for the

Fig. 5.—Posterior view of the ankle joint, all structures removed except the bandlike ligaments. Note the interosseous membrane, its fibers passing downward from the tibia to the fibula, the similarly directed fibers of the posterior inferior tibiofibular ligament; the middle band of the external lateral (fibulocalcanean) ligament; the posterior band of the external lateral ligament (fibuloastragalar ligament) attached to the lateral tubercle of the astragalus; the posterior fibers of the internal lateral ligament, and the posterior surface of the internal malleolus grooved for the tendon of the tibialis posticus. (From a preparation in the laboratory of operative surgery, University of Pennsylvania.)

reason that the fibula is already closely applied to the tibia at the point described. There is barely room to insert the blade of a scalpel between the bones at a point 3 inches (7.5 cm.), or less, above the tip of the fibula.

It was the particular merit of Dupuytren to systematize the teaching of various pioneers, such as Pott (1769), Bazille [10] (1771), Bromfeild [11] (1773) and Pouteau [12] (1783), who had recognized, as fractures, lesions which had been regarded by former generations as dislocations; and it is to Dupuytren's memoir on "The Fracture of the Lower End of the Fibula"[13] that surgery is principally indebted for its understanding of all these ankle fractures. It is true that Gosselin (1872) reproached Dupuytren with having propounded his theories of mechanism merely by means of his reasoning, and not on a basis of clinical observations or cadaveric experiments (though Dupuytren made a number of the latter, and presented records of 207 patients with ankle fractures). But there is much justification for Dupuytren's [14] statement that the mechanism detailed by patients cannot be relied on, since it is well known that in dislocations of the shoulder, for instance, they all will say the injury resulted from a fall on the point of the shoulder (because that is where they feel the pain), whereas the state of their elbow or hand proves quite the contrary. Similarly, I have known a patient with an ankle fracture badly united in abduction (fibular flexion) to assert that it was produced by adduction (tibial flexion), because her heel was prominent beneath the internal malleolus just after the accident; not realizing that the heel had been brought into that position by outward displacement of the point of the foot, the astragalus turning around the long axis of the leg.

Now it is to be noted that Dupuytren, who commended Pott for his accurate delineation of the typical fracture of the lower end of the fibula, was evidently of the belief that the typical fracture which he himself was describing was the same as that of Pott; and that this is still the opinion of the French is manifest from a footnote of

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10. Bazille: Mém. sur les sujets proposés pour le prix de l'Acad. Roy. de Chir., Paris **4**:563, 1778.

11. Bromfeild: Chirurgical Observations and Cases, London **2**:78, 1773.

12. Pouteau: Oeuvres posthumes, Paris **2**:267, 1783.

13. This memoir was said by Nélaton (Eléments de path. chir., Paris **1**:810, 1844) to have been read by Dupuytren in 1813 before the Académie de sciences. However, I have searched the "Procès-verbaux des séances de l'Académie des sciences" (Paris **5**:1812-1815) and find no reference to such an event; and Quénu (Rev. de Chir. **45**:5 (Footnote 2) 1912) states that he has had the original records of the Academy searched page by page, without finding any trace of Dupuytren's Memoir; and says that "until further information" he will consider as "mémoire princeps" the appearance of the essay in the "Annuaire medico-chirurgicale des hôpitaux et hospices civiles de Paris," 1819, p. 1. The essay is most easily accessible where first reprinted, in the second edition of Dupuytren's "Leçons orales" (Paris **1**:275, 1839).

14. Dupuytren: Leçons orales de clinique chir., Ed. 2, Paris **1**:328, 1839.

Quénu,[15] in which he says that the French mean by Dupuytren's fracture precisely what the English mean by Pott's. So that it is somewhat confusing to have J. Hutchinson, Jr.,[16] and some recent writers name and illustrate as Dupuytren's fracture a lesion which Dupuytren [17] encountered only once in more than 200 cases, and which consisted in a fracture of the fibula, rupture of the tibiofibular ligaments, and displacement upward of the astragalus along the fibular side of the tibia. As it is a fact that the fracture at the ankle most

Fig. 6.—Frontal section of the ankle joint. Adduction causes tension on external lateral ligament and if forced will tear off the external malleolus.

often seen, and therefore the most typical fracture, is that first described accurately by Maisonneuve (1840),[18] it is safe to assume that both

15. Quénu: Rev. de chir. **46**:367 (Footnote 2) 1912.

16. Hutchinson: Tr. Path. Soc., London **39**:238, 1887-1888.

17. Dupuytren: Footnote 14, p. 368.

18. Though, unfortunately, it is not known by his name, which is attached to a fracture produced experimentally by him on the cadaver, but which he never saw clinically. The most frequent and most typical fracture the French now call the "low Dupuytren" to distinguish it from the true Dupuytren or Pott fracture, which they name "Dupuytren type."

Pott's and Dupuytren's descriptions applied to this fracture and not to the rather hypothetic type they thought they were describing.

However, there is a typical, though rare, fracture at the ankle which corresponds closely enough to the original illustration of Pott to make it worthy of being called by his name; it is a typical "flexion fracture" (Biegungsbruch) of the fibula, usually 8 cm. (3¼ inches) or *higher* above the tip of the external malleous, accompanied by fracture of the internal malleolus and almost invariably by rupture of the inferior

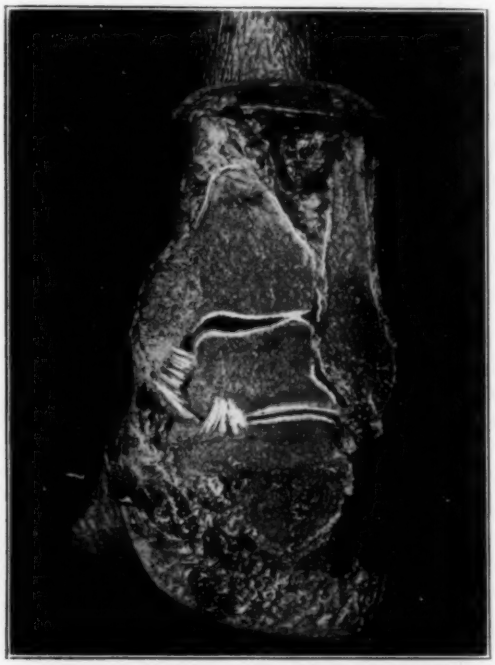

Fig. 7.—Frontal section of the ankle joint. Abduction causes tension on internal lateral ligament and will rupture this or fracture the internal malleolus before outward pressure on the external malleolus will fracture the latter or cause diastasis between tibia and fibula. (This specimen and that shown in Figure 6 are from a preparation in the laboratory of operative surgery, University of Pennsylvania.)

tibiofibular ligaments, permitting a diastasis between tibia and fibula. It is such a fracture as is illustrated in Fgure 26; and may well be called Pott's fracture, even though Pott in his own description placed the seat of the fibular fracture too low, and ignored the fracture of the internal malleolus and the tibiofibular diastasis.

Cooper [3] (1822) did not get away from the idea of the paramount importance of the dislocation of the tibia in these lesions (nor, it may

be remarked, did Malgaigne, Hamilton, Treves, or Stimson, many years later); and he described the fractures as mere incidents. But any modern surgeon will find it difficult to bring forth as extensive a list of severe ankle lesions as that recorded by Cooper, though he can easily excel Cooper in the number of lesions which (owing to the absence of deformity) can be certainly recognized only with the aid of the roentgen ray.

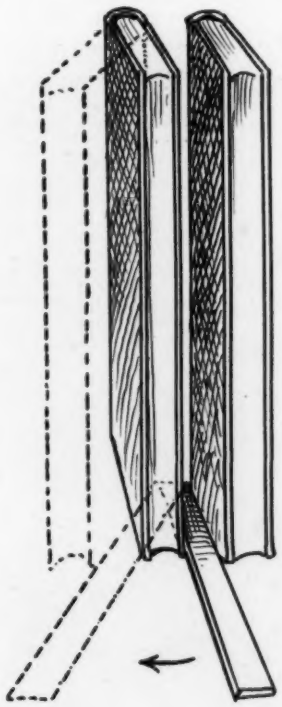

Fig. 8.—An illustration of Maisonneuve's explanation of fracture of the external malleolus by outward rotation of the foot around the axis of the leg. The books represent the malleoli and the ruler represents the foot. See page 100.

Cooper describes succinctly, but accurately, the following main groups of lesions; and scarcely one additional type has been discovered since, even with the aid of roentgenography:

1. *Simple Dislocation of the Tibia Inward.*—In this case there is a fracture of the fibula, two inches above its tip, carrying with it an attached fragment of the tibia;[19] the lower end of the upper fragment of the fibula rests on top of the

19. This is the "intermediate fragment" known to the French as the "third fragment of Tillaux," who produced it frequently in his cadaveric experiments.

astragalus,[20] and the tibia with the internal malleolus intact descends on the median surface of the astragalus. (This is illustrated in Figure 1 of Plate 16 of Cooper's monograph, first edition, 1822, and is reproduced here as Figure 2. The figure was copied by Vidal de Cassis[21] in his second edition [1846] as Figure 53 and the lesion was for many years known in France by his name; until Quénu recently called attention to the fact, quite clearly stated by Vidal, that the illustration was copied from Cooper.) This, says Cooper, is the most frequent of the dislocations of the ankle. It corresponds probably to the form now known to the French as the "low Dupuytren" fracture, though in the latter type, which is very frequent, there is very seldom any intermediate fragment detached from the tibia.

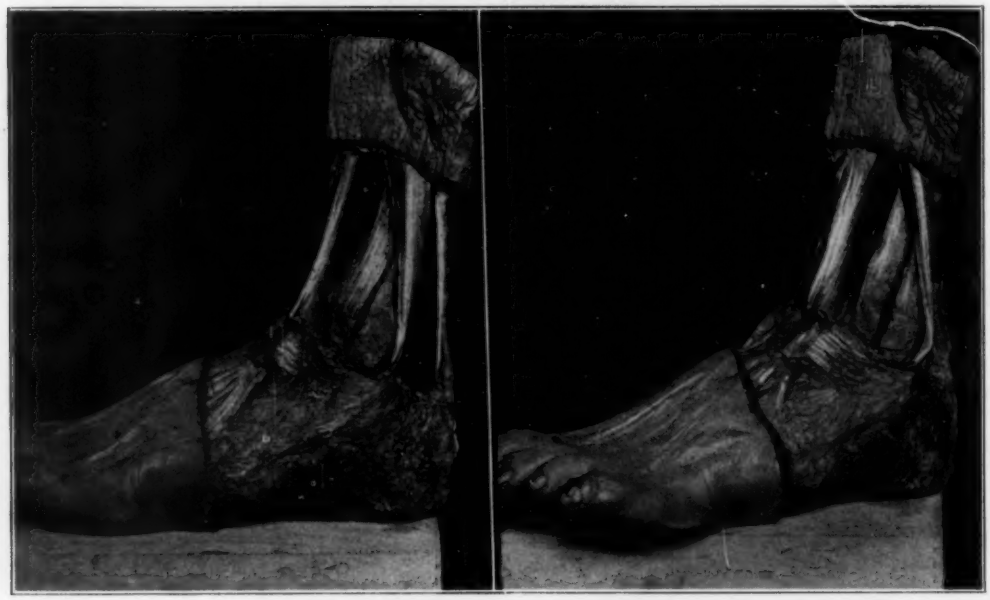

Fig. 9.—Oblique fracture of the fibula through the inferior tibiofibular joint, produced by an osteotome: in the first figure the foot is in the anatomic position; in the second it has been rotated outward around the axis of the leg, which is the only motion that will cause separation of the fragments. See page 101.

2. *Simple Dislocation of the Tibia Forward.*—In this case there is a fracture of the fibula, three inches above its tip; the internal lateral ligament is partly lacerated; the tibia and upper fragment of the fibula advance forward, and the tibia rests on the upper surface of the scaphoid and internal cuneiform. In

20. Richet (Unión méd. **20**:142, 1875) described such a case in which the astragalus was penetrated by the upper fragment of the fibula. Roentgenograms in anteroposterior view often show such an appearance, but the lateral views I have seen have always demonstrated that the fibula was behind, or very rarely in front of, the astragalus. Richet's lesion was demonstrated at necropsy.

21. Vidal de Cassis: Traité de pathologie externe, Ed. 2, Paris **2**:394, 1846.

partial dislocation of the tibia forward, the articular surface of the tibia is divided in two: the anterior part rests on the scaphoid and the posterior on the astragalus. The fracture of the fibula (as shown in Plate 17, Figs. 1 and 2, of Cooper's monograph, reproduced here as Figure 3) runs obliquely up and back, through the inferior tibiofibular joint. Thus there is here accurately described the posterior marginal fragment of the tibia, which many recent writers think they were themselves the first to discover after the introduction of roentgenography (Destot, Chaput, Cotton, Sear, etc.).

3. *Simple Dislocation of the Tibia Outward.*—This, says Cooper, is the most dangerous of the three, for it is produced by greater violence, etc. The internal

Fig. 10.—Mixed oblique fracture of the lower end of the fibula: the first lesion resulting from external rotation (A, I); occurs as isolated lesion in 25 per cent. of all cases of fracture of the ankle; as combined lesion, in 61 per cent. Usually invisible in anteroposterior views.

malleolus is obliquely fractured and separated from the shaft of the bone; the fractured portion sometimes consists only of the malleolus; at other times, the fracture passes obliquely through the articular surface of the tibia, which is thrown forward and outward on the astragalus before the external malleolus. The astragalus is sometimes fractured, and the lower extremity of the fibula is broken into several splinters. The internal and external lateral ligaments are usually intact; but if the fibula is not broken, the external lateral ligaments are ruptured. (Note that there is here described the "fracture by adduction" with a splitting off of a greater or less portion of the medial surface of the tibia—illustrated in Figure 2, from Cooper—a lesion which Tillaux thought had not before his time been observed by any surgeon).

Under the heading "Fractures of the Tibia and Fibula Near the Ankle Joint" (p. 353), Cooper describes a fracture of the fibula from two to three inches above the ankle joint (that is, not at a point from two to three inches above the tip of the external malleolus, but at the point where occurs the fracture called by Pott's name), produced by falling laterally while the foot is confined in a deep cleft; and a fracture of the tibia, in which the fracture line runs either obliquely down and in, toward the internal malleolus (i. e., the

Fig. 11.—Mixed oblique fracture of the fibula, with great obliquity. In these fractures, the obliquity may be great or slight; but the line of fracture is always higher on the posterior than on the anterior border of the fibula, and in 84 per cent. of the cases, the anterior end of the fracture line is between the tip of the malleolus below and the level of the articular surface of the tibia above. In 8 per cent. of the cases, the anterior end of the fracture line passes through the fibula at the level of the anterior tubercle of the tibia, and in 8 per cent. its anterior end is above this level (in which circumstances the tubercle is detached or there is diastasis). All tracings here reproduced were made directly from the roentgenograms.

ordinary spiral fracture of the shaft low down) or obliquely from 1 or 2 inches above the internal malleolus down and out into the ankle joint (i. e., the ordinary splitting fracture of the median part of the articular surface, usually dependent on a primary fracture of the fibula, and forming a less advanced degree of the fracture by adduction already described by Cooper as an outward dislocation of the tibia).

Fig. 12.—Mixed oblique fracture of fibula, with fracture of internal malleolus (A, II, b). The fibular fracture is visible only in the anteroposterior view (rare). Note that there is considerable lateral displacement of the astragalus, but that the posterior displacement is only apparent, not real, being due to the external rotation of the foot.

After Cooper, Maisonneuve [4] (1840). In a most remarkable essay, which has been ignored by most subsequent writers, Maisonneuve threw more light on the subject of ankle fractures than has any one since.

Up to that time, as he says, two theories prevailed to explain the mechanism of fractures of the lower end of the fibula: (1) by adduction, by which means the tip of the external malleolus was torn off; this was thought by Dupuytren

to be the most frequent variety,[22] and (2) by abduction, when, from pressure upward and laterally by the astragalus on the external malleolus, the latter was forced outward until the fibula broke at its weakest point, namely, entirely above the malleolus, that is, in the region described by Pott; this fracture being usually accompanied by a secondarily produced fracture of the internal malleolus or rupture of the internal lateral ligament. The second variety was thought by everybody, except Dupuytren, to be the most frequent.

Now Maisonneuve proposed another mechanism, which he believed explained the production of the most common type of fracture: this was simple deviation of the point of the foot outward, that is, external rotation of the foot in the

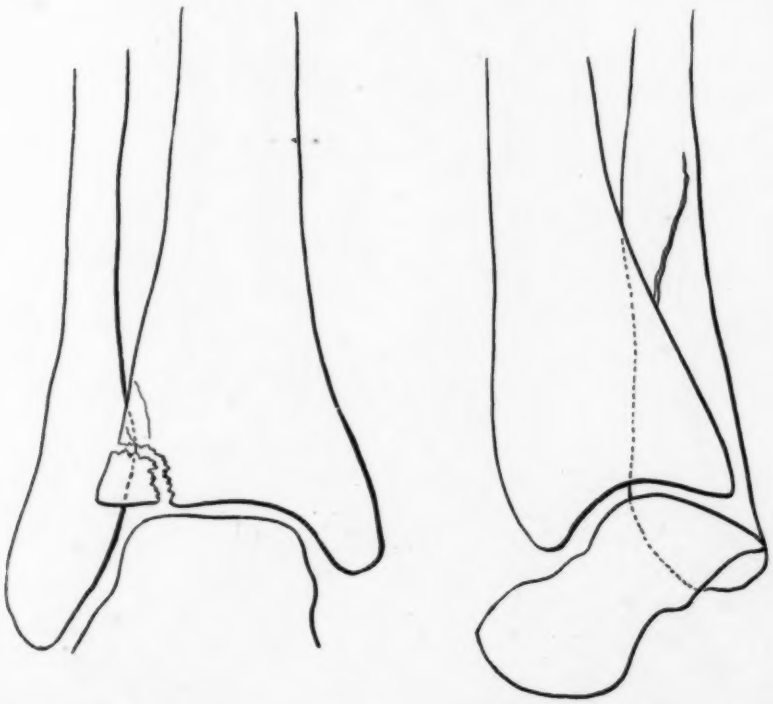

Fig. 13.—Mixed oblique (incomplete) fracture of fibula, complicated by, and subsequent to, avulsion of the anterior tubercle of tibia.

22. Dupuytren contended that the usual fracture which he described (and which he thought corresponded to that shown in Pott's illustration) was produced by a primary tearing off of the external malleolus when the foot turned so that the patient stepped on the outer edge of the sole; and that the rupture of the internal lateral ligament or fracture of the internal malleolus was produced secondarily by the patient's attempts to walk: whereupon, the external malleolus being already broken, the foot was forced into valgus; but he stated that when fracture of the fibula followed a turning outward of the foot, the internal malleolus broke first and the fibula only secondarily (Footnote 14, p. 327).

tibiofibular mortise around a vertical axis. In one recent necropsy, he had found an oblique fracture of the fibula, the line of fracture beginning on the anterior surface of the external malleolus 4 cm. above its tip and extending upward and backward to a point on the posterior surface of the fibula 8 cm. above the tip of the external malleolus. And he found that precisely this fracture was readily produced in the cadaver by the mechanism of outward rotation as above mentioned, whenever the tibiofibular ligaments held firm.

So far as I can ascertain, Maisonneuve was the first, and I might say almost the only, writer up to the present day to appreciate properly

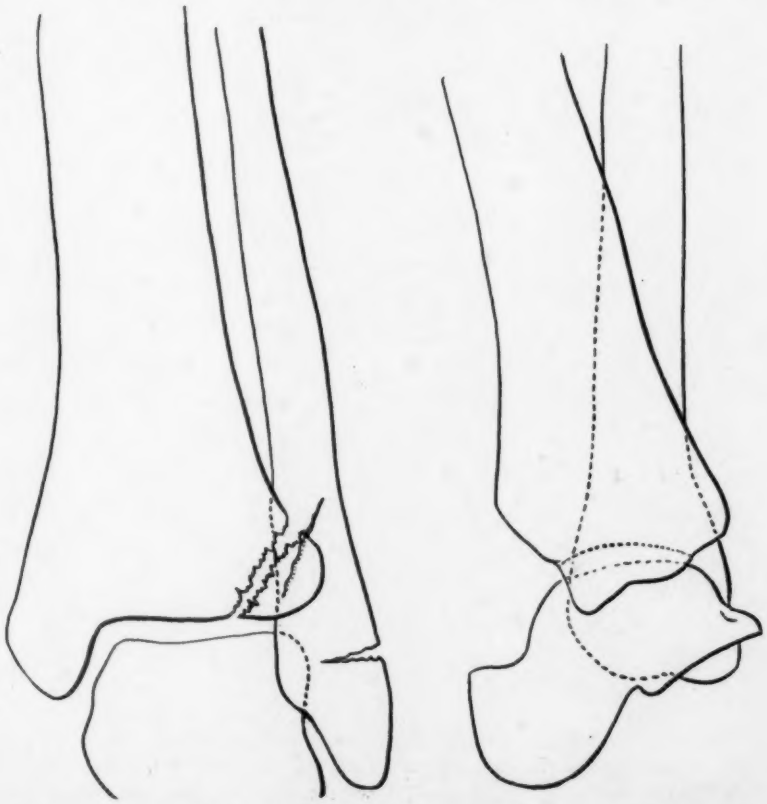

Fig. 14.—Mixed oblique (incomplete) fracture of fibula complicated by, and subsequent to, avulsion of anterior tubercle of tibia.

the importance of the inferior tibiofibular ligaments in the mechanism and classification of ankle fractures; and to recognize the great frequency of the oblique fracture of the fibula. This fracture, he found, was always the first lesion following external rotation of the foot, and occurred without any ligamentous injury. If the external rotation of the foot were continued far enough, the internal lateral ligament would rupture, or often the internal malleolus would be pulled off.

The resulting deformity, he found, was precisely the picture drawn by Dupuytren (*"coup de hache,"* etc.); and all of these phenomena disappeared when the position of the foot was corrected. If the inferior tibiofibular ligaments break, during this external rotation of the foot around the long axis of the leg, there occurs a greater or less diastasis of the inferior tibiofibular joint; and if the movement still continues, the fibula breaks not at the usual site, but in its upper third, or at least in its middle third. It is to this fracture of the fibula in its upper third, produced after diastasis of the inferior tibiofibular joint has occurred,

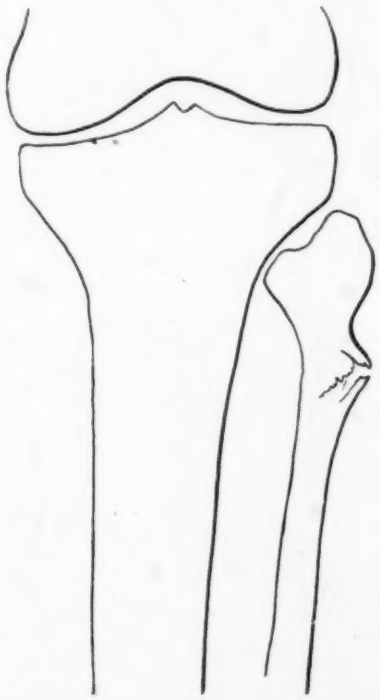

Fig. 15.—Incomplete fracture of fibula below its head (A, I, Variant), apparently from compression in its long axis (see pp. 103 and 107). No bone lesion at ankle. There was a history of a fall down three steps while carrying a load of 100 kilograms on the back. Injury occurred so quickly the patient did not know whether he turned his ankle or struck the side of his leg against the marble steps. Pain was so great that he had to be brought to the hospital by a patrol wagon. Examination showed fracture in the upper third of the left fibula (crepitus and deformity). The ankle was swollen over the external malleolus.

that the name of Maisonneuve's fracture has been attached; though he never encountered such a case clinically.

Maisonneuve's chief contribution was his recognition of the oblique fracture of the lower end of the fibula as the first stage of a lesion

frequently including also a fracture of the internal malleolus or a rupture of the internal lateral ligament; and it is a pity that his name is not applied to this very frequent fracture rather than to one of the utmost rarity. Moreover, he was no doubt correct, as I have previously intimated, in believing that this was the lesion whose clinical signs were described by Dupuytren, and that the latter erroneously thought these

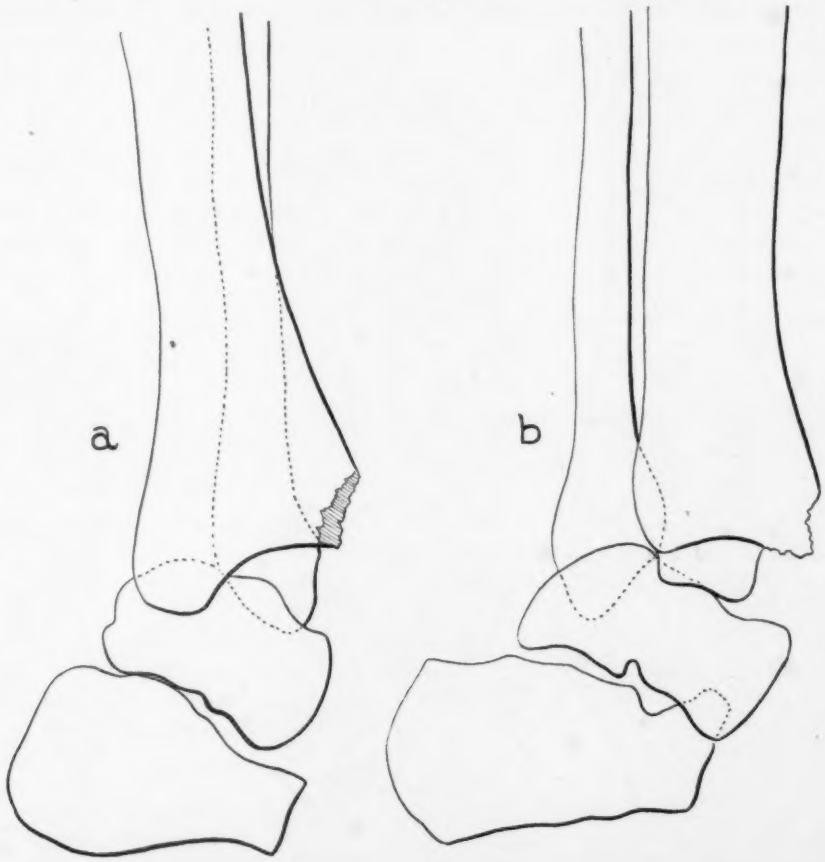

Fig. 16.—Diastasis with displacement of fibula behind tibia. The anterior tubercle of tibia has been torn off and accompanies the fibula, which is not fractured. Diastasis by external rotation (mechanism described by Huguier[49]): (*a*) anteroposterior view, and (*b*) lateral view before reduction. See also Figure 17 (A, I, complication) and page 103.

cases were such as had been depicted by Pott. Maisonneuve's chief, and almost his only, error, as it seems to me, was his failure to appreciate that a lesion did exist, consisting of a fracture well above the inferior tibiofibular joint, associated with fracture of the internal malleolus

or rupture of the internal lateral ligament, and that this type of fracture, though rare, was yet much less rare than a fracture of the fibula in its upper third or even in its middle third.

After Maisonneuve, Tillaux [5] (1872). Tillaux did not distinguish in his cadaveric experiments between abduction (fibular flexion) of the foot and the same movement combined with slight outward rotation (i. e., around the long axis of the leg). Abduction, he found, caused (*a*) rupture of the internal lateral ligament or tearing off of the internal

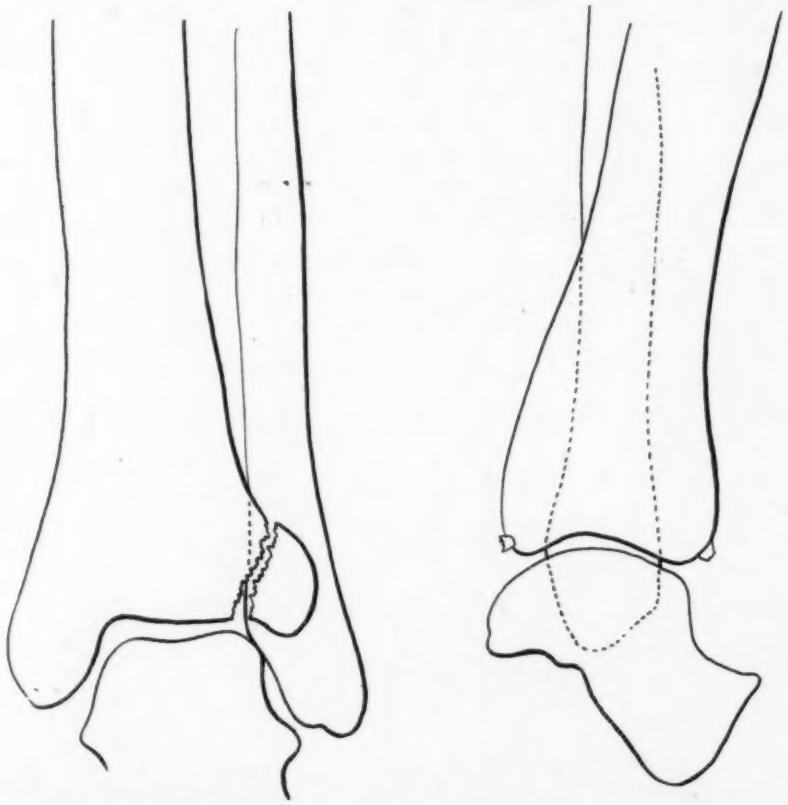

Fig. 17.—Same case as that shown in Figure 16, after reduction. Fracture of anterior tubercle of tibia clearly seen.

malleolus, or (*b*) the same lesions plus fracture of the fibula above the inferior tibiofibular ligaments, or 6 to 7 cm. above its tip; the tibiofibular ligaments may not rupture, but the degree of rupture of these ligaments determined the degree of the displacement; and he found the fibula could not be broken in abduction movements unless the internal malleolus or the internal lateral ligament had been previously broken. He also called particular attention to the great frequency of a fragment

of bone torn off the lateral border of the tibia by the inferior tibiofibular ligaments; this fragment has since been known to the French as the "third fragment of Tillaux" (remember it had been described and illustrated by Cooper and by Vidal de Cassis).[23]

It is thus seen that while Maisonneuve minimized the mechanism of pure abduction, Tillaux magnified it out of all reason; for the fact remains that the vast majority of fractures are not of the type produced experimentally by Tillaux by abduction (and which correspond rather to the original type with which Pott and Dupuytren thought they had to deal), but are of the type produced experimentally by Maisonneuve by external rotation. In his description of the results of adduction (tibial flexion), Tillaux agreed with his predecessors and successors (the results are noted below under the account of Hönigschmeid's experiments); but he also describes a rare result of adduction which consists in a transverse supramalleolar fracture of the tibia sometimes occurring in those cases in which the fibula breaks above the inferior tibiofibular joint instead of below it (Fig. 43). This mechanism, he says, involves great strain on the superior tibiofibular joint until the fibula breaks above its malleolus; and he observed a case in life in which this transverse supramalleolar fracture (of the tibia only) was complicated by a diastasis of the *upper* tibiofibular joint. (In this connection, one of the specimens from the Mütter Museum [72] is of much interest.)

Hönigschmied [6] (1877), as already remarked, made 125 experiments on the cadaver, to determine the mechanism of ankle fractures, and

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23. Souligoux (Bull. et mém. Soc. de chir. de Paris **38**:1103, 1912) described two unpublished diagrams made by Tillaux to illustrate this "third fragment:" the anterior view showed the anterior tubercle of the tibia torn off, while the posterior view showed the posterior tubercle torn off. Apparently, then, it was Tillaux's opinion that either tubercle could represent his third fragment. A larger fragment from the inferior lateral margin of the tibia may exist, known to the French as the "intermediate fragment of Verneuil," but it is quite rare. This seems to correspond to what Roberts and Kelly (the advance proof sheets of whose second edition, 1921, Dr. Roberts has very courteously sent me) describe as the "drunkard's fracture." Confusion is added to this matter by the statement of Thaon (Bull. Soc. anat. de Paris **45**:212, 1870), in presenting a necropsy specimen of a posterior marginal fracture, that he had often seen this posterior fragment in experiments made by Tillaux; and by the statements of Demoulin (Bull. et mém. Soc. de chir. de Paris **38**:1103, 1912), of Mauclaire (Bull. et mém. Soc. de chir. de Paris **38**:1141, 1912), and of Viallet (Rev. de chir. **46**: 690, 1912) that the third fragment of Tillaux may easily be mistaken in roentgenograms for a posterior marginal fragment. My own belief is that the third fragment of Tillaux is the anterior tibial tubercle and that the fragment composed of the posterior tubercle and that known as the posterior marginal fragment usually are indistinguishable.

a brief summary of them is given, as it will be necessary to refer to them frequently in the latter portions of this memoir.

1. *Plantar hyperflexion, twenty experiments.* In fourteen cases he got rupture of the internal lateral ligament and anterior fibers of the external lateral ligament; the rupture sometimes occurred from the tarsus, sometimes from the leg bones; sometimes a small fragment of bone was torn loose. In five cases (aged subjects), one or both of the malleoli were broken.

2. *Dorsal hyperflexion, twenty-one experiments,* always with previous division of the tendon of Achilles. In seventeen the internal malleolus was broken, by push of the tarsus against its tip. In two the internal lateral ligament was ruptured. In two the only lesion was in the tarsus.

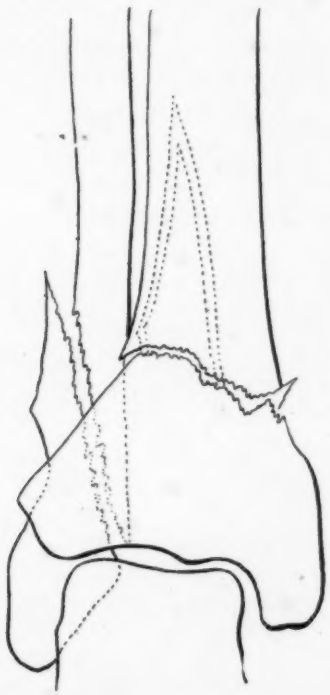

Fig. 18.—Mixed oblique fracture of fibula, internal malleolus represented by whole lower end of tibia (A, III).

3. *Tibial flexion (adduction or supination), seventeen experiments.* In all the external malleolus (five) or external lateral ligament (twelve) was broken. He never got fracture of the internal malleolus or of the tibia.

4. *Fibular flexion (abduction or pronation), twenty-two experiments.* In fifteen the internal lateral ligament or the internal malleolus broke. In seven the only lesions were in the tarsal (two) or subastragalar (five) ligaments. He never obtained a primary fracture of the external malleolus: in two cases only did it break, the lesion being a compression fracture at its tip. (If the inferior tibiofibular ligaments had previously ruptured, the weight of the patient's body, in a fall, would have fractured the fibula above these ligaments.)

5. *Inward rotation of foot around long axis of leg, twenty experiments.* In nineteen there were lesions of the ligaments (fifteen involved the tarsal ligaments, of which six involved also the external lateral ligament; while in four the external lateral ligament was alone involved). In one the anterior margin of the external malleolus was broken, evidently being torn off by the anterior fibers of the external lateral ligament. (Such a fracture had been described by Wagstaffe,[24] in 1875, and was later studied by L. LeFort[25] (1886) and by his pupil LeRoy[26] (1887).

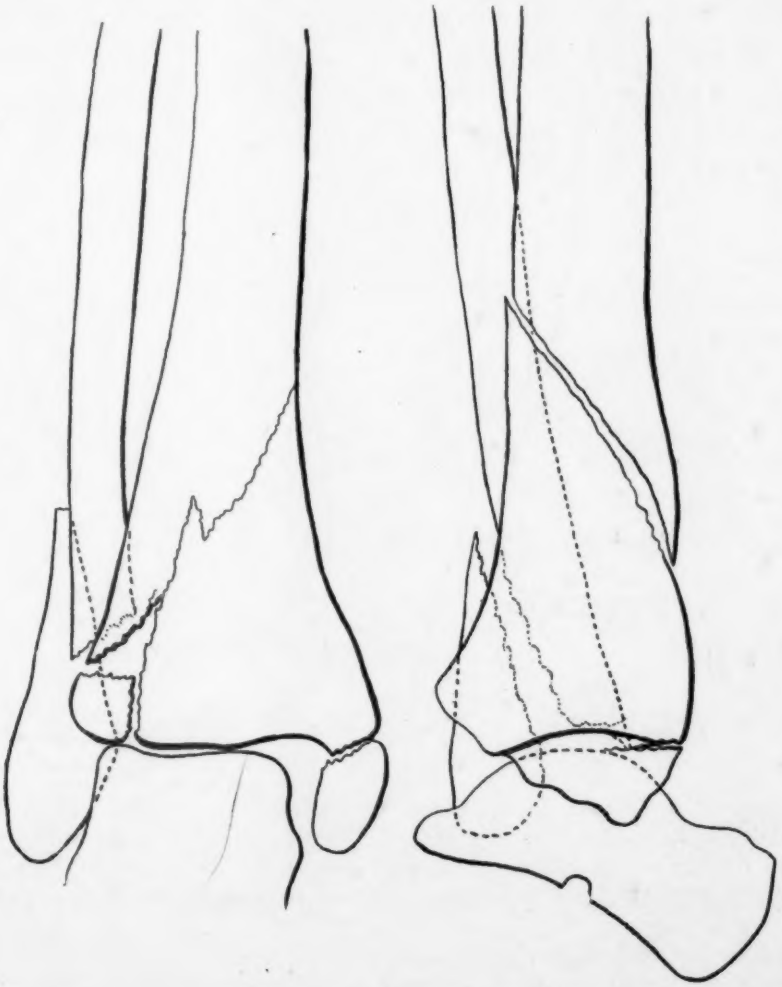

Fig. 19.—Mixed oblique fracture of fibula, with fracture of internal malleolus and of whole lower end of tibia. There is also a fracture of the anterior tubercle of the tibia (A, III).

24. Wagstaffe: Saint Thomas's Hospital Reports, London **6**:43, 1875.

25. LeFort: Bull. gén. de Thérap., Paris **110**:193, 1886.

26. LeRoy: De la fracture marginale antérieure de la malleole externe. Paris, 1887.

6. Outward rotation of foot around long axis of leg, twenty-two experiments.

(1) In twenty, fracture of the lower end of the fibula occurred: (*a*) in fourteen of these there was no diastasis of the inferior tibiofibular joint; in two there was rupture of the external lateral ligament with sprain fracture of the tip of the external malleolus. In seven there was only slight obliquity of the fracture line, upward and backward (four had no other lesion, three had also rupture of the internal lateral ligament). In five the line of fracture was distinctly oblique, involving more of the posterior surface of the fibula (two of these five had also rupture of the internal lateral ligament). (*b*) In six cases there was diastasis, usually detaching a small intermediate fragment from

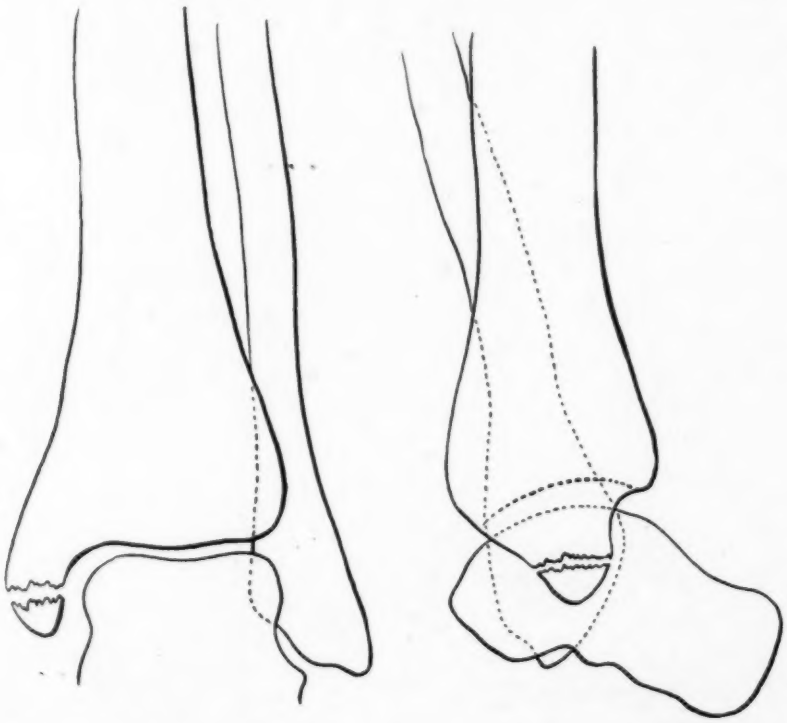

Fig. 20.—Abduction fracture, first degree (B, I): avulsion of internal malleolus. In this patient the internal malleolus extended abnormally low. This fracture occurs in 6.6 per cent. of all fractures at the ankle. All forms of the abduction type together comprise about 21 per cent. of the cases.

the tibia, and with rupture of the internal lateral ligament or fracture of the internal malleolus; in four of these six cases the fracture of the fibula was oblique upward and backward, and in the remaining two cases the fibula was broken obliquely in its upper third (i.e., Maisonneuve's fracture).

(2) In two cases diastasis occurred without fracture of the fibula.

Hönigschmied concluded that these oblique fractures of the lower end of the fibula produced during external rotation are not due, as Maisonneuve taught, to the outward pressure of the astragalus on the anterior border of the external malleolus, but to the pull exerted by the posterior band of the external lateral ligament, which tears off the posterior portion of the malleolus.

Destot, in the thesis of Bondet [27] (1899) and in his own monograph [8] (1911), attempted a physiologic classification, abandoning that based on the mechanism. He pointed out that the main function of the tibia was that of support, while the fibula acted merely as a splint along the outer side of the ankle joint to maintain the direction of the foot. Thus his classification embraces (1) those fractures which involve only the mortise, and disturb the equilibrium of the foot; and (2) those

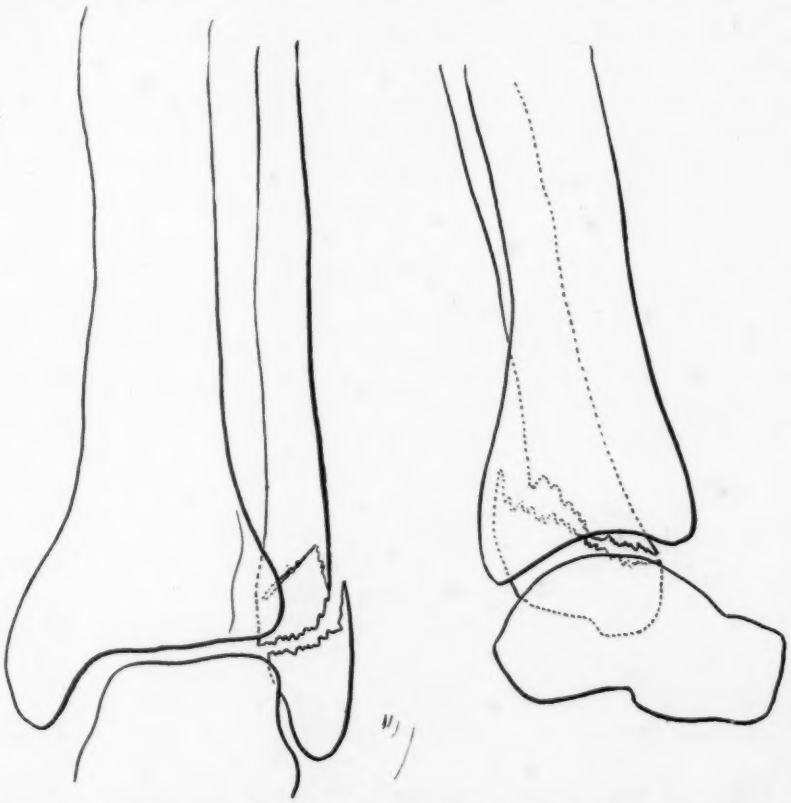

Fig. 21.—Fracture of fibula below inferior tibiofibular ligaments, with very slight obliquity, accompanied by rupture of the internal lateral ligament, as evidenced by lateral displacement of astragalus. This type of fracture is intermediate between the fracture by external rotation already discussed and the fracture by abduction. It is impossible from the roentgenogram alone to say to which of the two types such a fracture really belongs (A, I or B, I).

which involve the "pilon tibial" (the tibial "pestle") and hence compromise the support of the body. Quénu [28] (1912) addressed very severe criticisms against this classification, and, pointing out that the

27. Bondet: Thèse de Lyon, 1899.
28. Quénu: Rev. de chir. **45**:1, 211, 416, 560, 1912.

gravity of the lesions depends entirely on whether they are isolated (involving one bone only) or associated (involving both tibia and fibula), erected on this basis an elaborate scheme which, as I have already said, is less a classification than a catalog.

Finally, Tanton [29] (1916) attempted to combine the merits of Destot's and of Quénu's classifications, with a reasonable degree of success. Indirect forced movements, he said, may involve (1) the malleolo-astragalar or tarsal ligaments (sprain); (2) the inferior tibiofibular ligaments (diastasis); (3) the malleoli (isolated fractures), very frequent; (4) the "pilon tibial" (isolated), very rare; (5) the malleoli and tibial *pilon* (frequent). Thus he tabulated the lesions as follows (omitting sprains and simple diastases):

I. Fractures of Malleoli—
  1. Isolated:
    (*a*) External malleolus.
    (*b*) Internal malleolus.
  2. Associated:
    (*a*) Low bimalleolar.
    (*b*) Low Dupuytren.
    (*c*) Typical Dupuytren (Pott's).
    (*d*) Maisonneuve.

II. Fractures of Pilon—

A. Partial.
  1. Isolated:
    (*a*) Anterior or posterior tubercles of tibia.
    (*b*) Marginal: anterior, external, posterior.
    (*c*) Wedge-shaped internal fractures.
  2. Associated:
    (*a*) Anterior margin with a malleolar fracture.
    (*b*) External margin with a malleolar fracture.
    (*c*) Posterior margin with a malleolar fracture.

B. Total (Eclatements).

Stimson,[7] both in his lecture of 1892, and in the last edition [30] (1917) of his book on "Fractures and Dislocations," makes a distinction between fractures in which the mechanism of fibular flexion (abduction) predominates and those in which outward deviation of the point of the foot (outward rotation) predominates.

In the former, he says, the internal malleolus is broken, followed by rupture of the inferior tibiofibular ligaments (or sometimes detachment of a piece of the tibia), and then occurs a fracture of the fibula "close above the malleolus;" while in the latter there occurs rupture first of the anterior inferior tibiofibular ligament, then of the anterior fibers of the internal lateral ligament.

29. Tanton: Fractures du membre inférieur, Paris, 1916.
30. Stimson: Fractures and Dislocations, Ed. 8, Philadelphia, 1917, p. 450.

and almost coincidentally the fibula breaks by the twisting of its lower end, the line of fracture being very oblique.

But in spite of all this comparatively explicit and positive teaching for so many generations, the fact is undeniable that the average surgeon knows little or nothing about ankle fractures; and that most teachers of surgery either leave their students with but a confused notion of the subject, or else teach them positively what is certainly not correct.

Fig. 22 (B, II, a).—Fracture of both malleoli by abduction. A heavy steel I-beam, lying on the ground beside the patient while he was standing at work was tipped over and struck the lateral surface of his leg, causing sudden abduction of the foot. He was not knocked down, but he felt a sudden pain in the ankle. He was able to walk on the leg. In the lateral view, the fracture of the external malleolus (which occurred subsequent to that of the internal malleolus, and was incomplete and subperiosteal) is not visible at all, while even that of the internal malleolus can hardly be seen.

These examples may be cited:

"Fracture of the lower end of the fibula is a very frequent injury, resulting from indirect violence, the foot, as a rule, being turned violently outward (abduction fracture); as the astragalus forces the external malleolus outward,

the tibiofibular ligaments act as a fulcrum, so that the fibula is bent in against the tibia above the attachment of these ligaments, and finally breaks at this point from five to eight cm. above the ankle joint; the internal malleolus often is avulsed from the tibia, at the same time; and to this combined lesion the name of Pott's fracture is given." (Ashhurst: Surgery, Philadelphia, 1914. p. 380.)

"What is a Pott's fracture? . . . It is a fracture of the lower end of the fibula that is produced by the foot turning outward when the injury is sustained. What happens when the foot turns outward? The external surface of the astragalus presses against the tip of the external malleolus, and the fibula

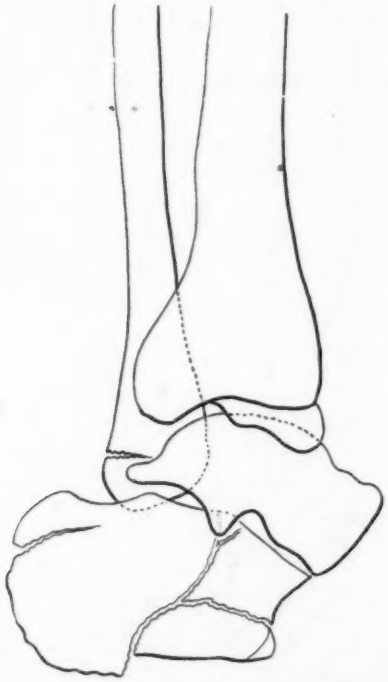

Fig. 23.—Crush fracture of calcaneum permitting fracture of external malleolus by compression. See page 99.

breaks primarily. If the interosseous ligament is strong, the fibula breaks within the first two and one-half inches above the tip of the external malleolus, otherwise only the tip of the fibula is broken off on a line with the tibio-astragaloid articulation, as in the case before us today. In a typical Pott's fracture the fibula fractures higher up.

"Next occurs a fracture of the internal malleolus, due to traction on the internal lateral ligament or rupture of the ligament. Then, as the pressure is continued with the foot everted and the weight of the patient's body is brought to bear on the foot, what happens? The astragalus is crowded up against the

tibiofibular articulation, and, acting as a wedge, forces apart the tibia and fibula and splits or tears the interosseous ligament."[31]

"The mechanism of Pott's fracture consists first in forcible eversion of the foot until the outer surface of the astragalus is driven against the fibular malleolus; secondly, in fracture of the shaft of the fibula, usually above the inferior tibiofibular joint, the unyielding interosseous ligament acting as a fulcrum; thirdly, by a continuation of the vulnerating force, the acute supero-external border of the astragalus develops as a wedge and is driven upward between the tibia and fibula until it springs the joint by lacerating the interosseous membrane."[32]

Cotton[33] (1910) describes all "eversion fractures" as Pott's fractures, and, though he points out the inaccuracy of this term, does not dwell particularly on their mechanism, merely quoting Stimson's account already given.

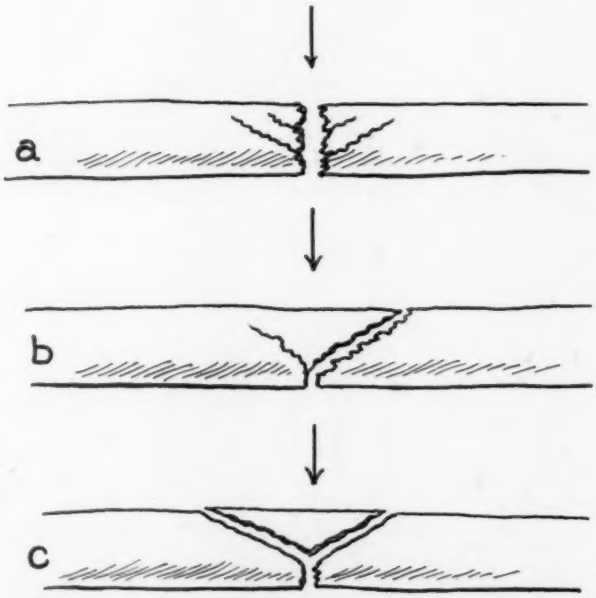

Fig. 24.—Messerer's diagram illustrating the mechanism of fracture by bending (Biegungsbruch) or flexion: (*a*) minute examination always shows in fractures apparently transverse delicate lines diverging from the main fracture and extending backward toward the surface of the bone which has been compressed or flexed, and away from the surface of extension; (*b*) an oblique fracture represents one of these diverging lines which has become a complete fracture; (*c*) very frequently a wedge-shaped fragment is detached, with its base on the compression (concave) surface and its apex toward that of extension (convex surface). See Figures 25, 26, 28, 29, 35, 36.

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31. Murphy: Surgical Clinics, Philadelphia **3**:2, 1914.
32. Murphy: Surgical Clinics, Philadelphia **1**:620, 1916.
33. Cotton: Dislocations and Joint Fractures, Philadelphia, 1910, p. 545.

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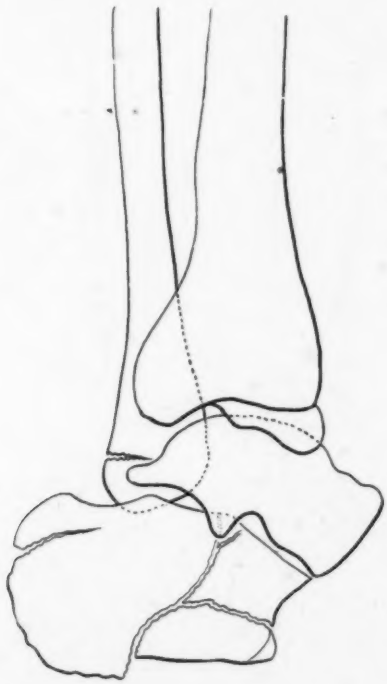

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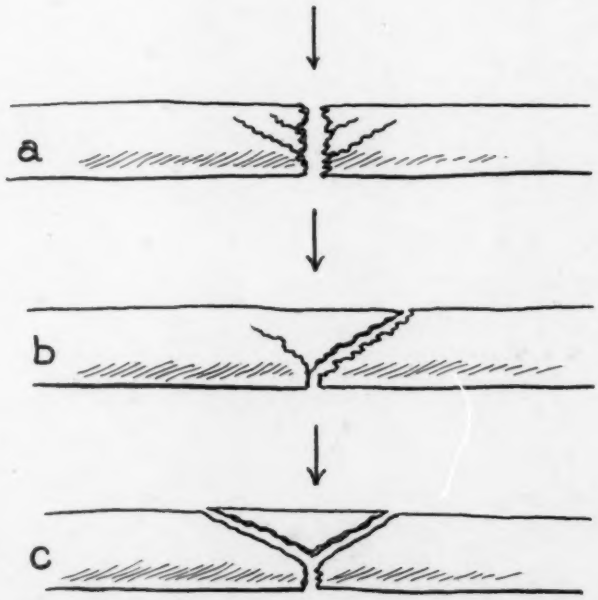

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31. Murphy: Surgical Clinics, Philadelphia **3**:2, 1914.
32. Murphy: Surgical Clinics, Philadelphia **1**:620, 1916.
33. Cotton: Dislocations and Joint Fractures, Philadelphia, 1910, p. 545.

Roberts and Kelly[34] (1916), in describing "fracture by eversion and abduction," say the mechanism is first fracture of the internal malleolus or rupture of the internal lateral ligament; "as the force continues the astragalus is forced against the external malleolus, fixing the latter, at the same time the weight of the body falling outward carries with it the leg and the fibula; the fibula is checked by the astragalus [Do they mean that the external malleolus is kept from moving inward as the body of the fibula moves outward?] and kept attached to the tibia by the tibiofibular ligament; either the latter ruptures or a line of fracture occurs at the weakest point of the fibula just above the point of attachment of the tibiofibular ligament. It is here that the fixed portion

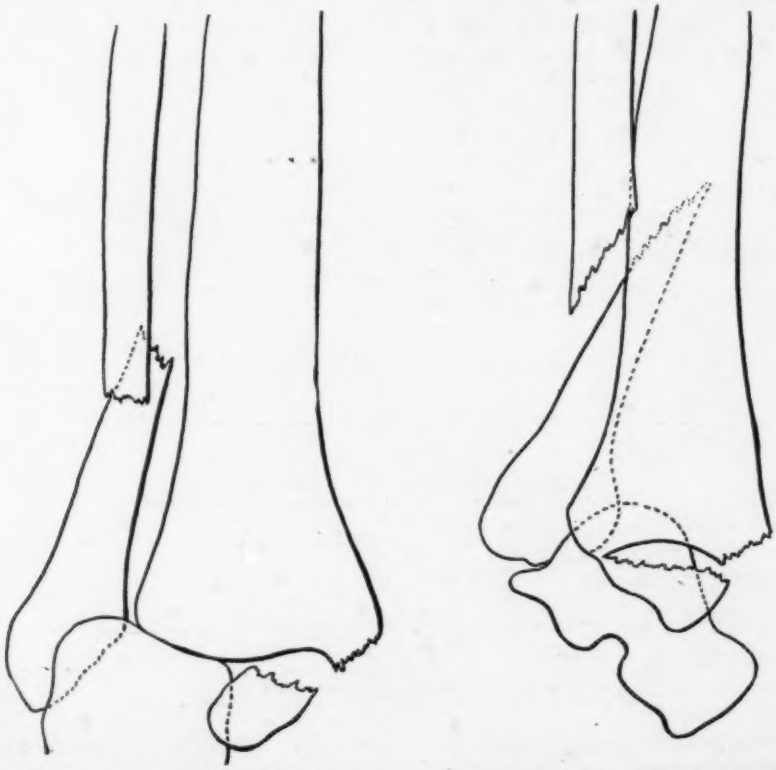

Fig. 25.—Fracture by abduction, second degree (B, II, b) (Pott's fracture, Dupuytren type): a primary fracture of the internal malleolus or rupture of the internal lateral ligament, followed by diastasis (with or without an intermediate fragment), finally succeeded by fracture of the fibula above the inferior tibiofibular joint by bending (Biegungsbruch).

of the fibula meets the potential moving upper portion, and fracture occurs about 2 to 2¼ inches above the malleolus. The line of fracture of the fibula depends to a great extent on whether the fall is directly outward, or whether some torsion of the tibiotarsal joint occurs at the moment of fracture."

34. Roberts and Kelly: Treatise on Fractures, Philadelphia, 1916, p. 580.

On page 590, they describe another (?) mechanism: "In addition to the mechanism described under fibular fracture, there is the effect due to the weight of the body being applied simultaneously with the violence causing the supramalleolar fracture of the fibula; by this force the outer trochlear surface of the astragalus is carried sharply against the outer portion of the articular surface of the tibia, by external rotation and abduction of the foot, so that either the tibiofibular ligament must give way or the outer edge of the tibia be broken away from the shaft. Dislocation upward of the astragalus between the tibia and fibula may occur."

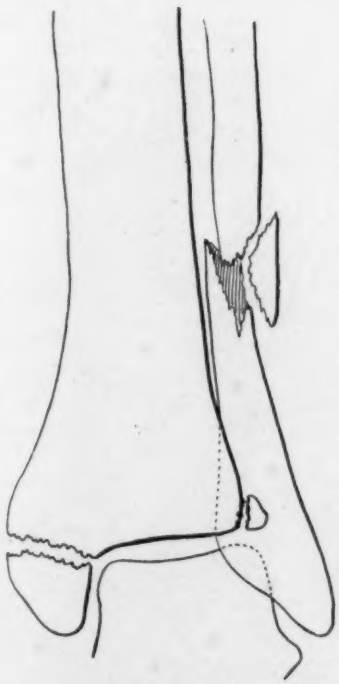

Fig. 26.—Fracture by abduction, second degree (B, II, b) (Pott's fracture, Dupuytren type): diastasis evidenced by detachment of fragment from anterior tubercle of tibia (intermediate fragment); fracture by bending clearly shown by detachment of wedge from concave side of fibula.

Rose and Carless[35] (1920) distinguish between Pott's fracture and Dupuytren's fracture thus:

"In Pott's fracture, sudden abduction, usually combined with eversion of the foot, results in severe strain on the internal lateral ligament, which gives way, or the base of the internal malleolus is torn off. The astragalus is at the same time driven outward against the external malleolus, and the force is thence transferred up the fibula, which bends and breaks at some weak spot. Generally, eversion is a large element in the force that produces the fracture which then runs obliquely from above downward and forward through the malleolus;

35. Rose and Carless: Manual of Surgery, Ed. 10, London, 1920, p. 632.

less frequently it is due to a pure abduction and may then be situated in the position originally described by Pott, viz., about three inches above the tip of the malleolus, and is transverse, the upper end of the lower fragment being displaced inward toward the tibia. The inferior interosseous ligament remains intact. . . .

"In Dupuytren's fracture a much more serious lesion is produced. The interosseous tibiofibular ligament yields more or less completely, or the flake of the tibia to which it is attached is torn off."

Speed[36] (1916) thus describes the mechanism of fractures about the ankle (p. 772): "When abduction and eversion of the foot are the cause of the fracture, the astragalus is pushed outward, and the fibula tends to break at a

Fig. 27.—Diastasis of inferior tibiofibular joint, following rupture of internal lateral ligament, but not succeeded by fracture of fibula (B, II, b, 1, Variant).

point above the termination [presumably he means the *upper* termination] of the tibiofibular ligament in a transverse or oblique line from compressive force. Coincidently the internal lateral ligament either ruptures, or holding its insertion into the tibia, pulls off the internal malleolus squarely near its lower end (Fig. 566). [This figure represents a "low Dupuytren" fracture.] If this eversion continues strongly, the lower fibular fragment may be separated a little from the tibia by tearing of the tibiofibular ligament, and the internal malleolus is correspondingly dragged outward by the internal lateral ligament,

36. Speed: Textbook on Fracture and Dislocations, Philadelphia, 1916, p. 772.

and comes to lie under the joint surface (Fig. 567). [This figure shows a Pott's fracture, with wide diastasis; and the latter certainly preceded the fracture of the fibula and did not, as Speed contends, follow it.] Some torsion is present in all these cases. . . . If the torsion is a more predominating feature in conjunction with the eversion, we obtain the spiral fractures of the external malleolus, as this point projects lower down than the internal malleolus and meets with most of the force in external torsion and eversion. [The reason alleged is insufficient explanation.] These spirals, in a quickly acting force, are above the lower end of the tibiofibular ligament which by a slight elasticity holds while the rigid bone gives; but in slower acting force with more eversion or compressive violence from the body weight, the extreme end

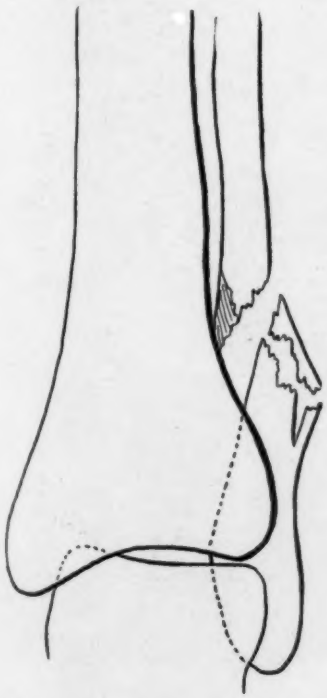

Fig. 28.—Fracture of lower fifth of fibula (surgical neck) by direct violence (kick of horse): there is no diastasis and under such circumstances the typical bending fracture (wedge detached from surface of flexion), which has been overcorrected by adduction of foot, could not have been produced by indirect violence (pressure of astragalus outward on external malleolus).

of the external malleolus is fractured and splintered up in a spiral manner. As a rule there is not much damage to the internal malleolus and the internal lateral ligament in this mechanism (see Fig. 568)."

This figure shows a low mixed oblique fracture of the fibula, unreduced, though the foot is in adduction and inward rotation. Speed says reduction has not been secured because of laceration of the external lateral ligament. But how could such a laceration occur in the mechanism he is discussing? Speed adds that "sometimes in eversion, in addition to fibular fracture, the tibiofibular ligament is torn, a condition permitting wide separation between the bone ends

and possibly accompanied by a shell of bone pulled out from the tibia." Writing (p. 785) of "malleolar fractures caused by inversion of the foot," he says, "The mechanism of isolated fractures of the internal malleolus is that of fall or compression from body weight against the talus, which is tipped inward by the inverted foot. . . . For this result the inversion must not be great, because the pull of the external lateral ligament would also pull off the external malleolus." (Thus he regards fracture of the external malleolus as a secondary and not the primary lesion in adduction fracture. But the illustration he gives as an instance of isolated fracture of the internal malleolus is not happily chosen [Fig. 592], as it shows the astragalus displaced from the external malleolus,

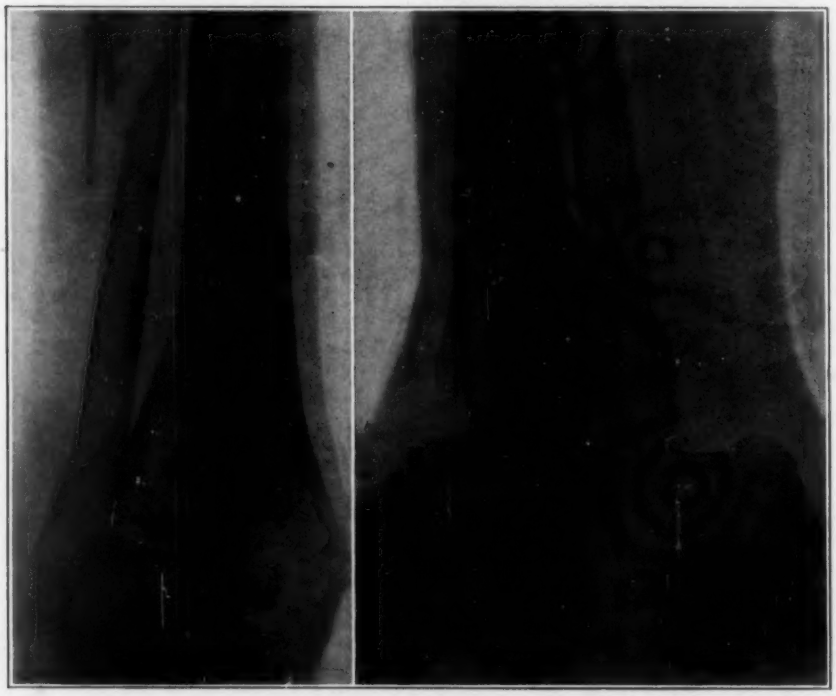

Fig. 29.—Fracture by abduction, third degree (B, III). The internal malleolus is represented by the whole lower end of the tibia; the fibula is broken by bending; there may or may not be an intermediate fragment. There were only two cases of this kind in our series of 300 cases.

an occurrence obviously permitted only by partial rupture of the anterior fibers of the external lateral ligament or sprain fracture of its tip.)

MECHANISM

Now, after the somewhat tedious historical review given above, it is worth while, before proceeding further in our inquiries, to pause a moment to refresh our knowledge concerning the structure and functions of the ankle. We have read of eversion, of abduction, of rotation,

of torsion, compression and bending, of malleolus and fibula, of ligaments and tubercles, of sprains, displacements and diastases; but it is clear that few authors use any of these terms in the same senses, and

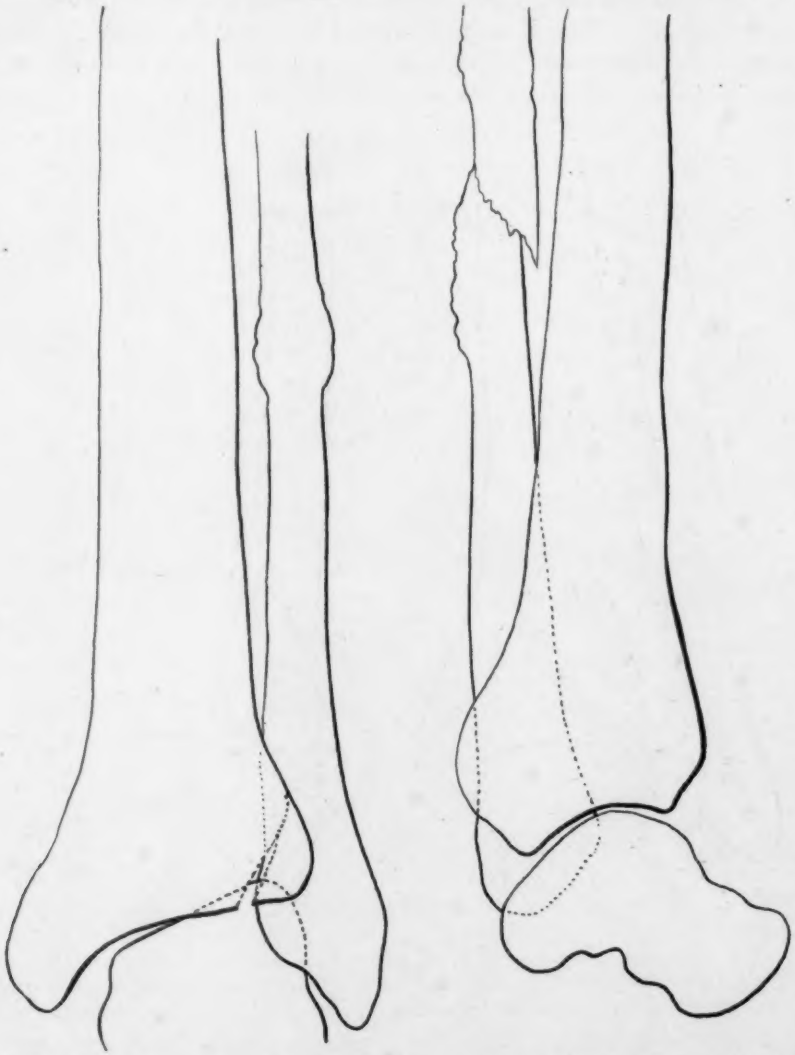

Fig. 30.—Fracture by abduction (B, II, b, 1): rupture of internal lateral ligament, followed by diastasis (note the intermediate fragment), and this succeeded by a bending fracture of fibula, in this case at an unusually high level.

that some of them probably do not know what they mean themselves. But what Nélaton [37] (1844) said of surgery in general may well be

37. Nélaton: Eléments de path. chir., Paris, 1844, vol. 1, Preface.

applied to this particular part of surgical knowledge: *"Rien en chirurgie n' est assez abstrait pour que l'obscurité s'excuse par le sujet même, et sur un grand nombre de points la science est assez avancée pour que la verité apparaisse au milieu des controverses à celui qui la cherche sans préoccupation."* And if what I succeed in explaining in the following pages appears to you to be nothing new, you will admit its truth; and I shall be satisfied. If on the other hand, you do not agree with my

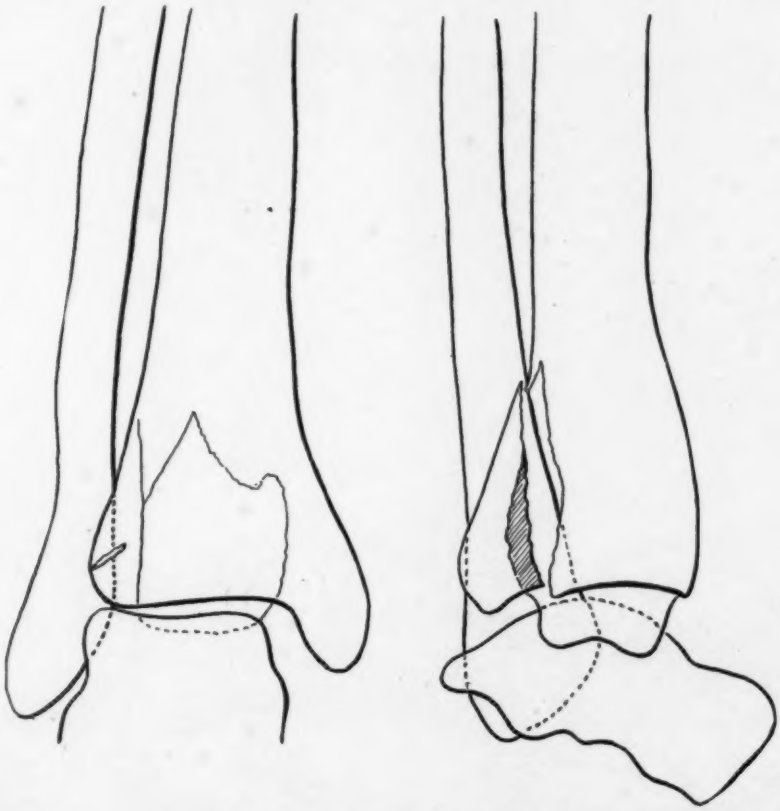

Fig. 31.—Fracture of posterior margin of tibia, associated with a tibiofibular diastasis, as evidenced by sprain fracture at this joint. The posterior marginal fragment which is unusually large is visible also in the anteroposterior view.

conclusions, that will not in the least disconcert me, for it will not impair their truth.

The ankle joint is formed above by the tibiofibular mortise, and below by the trochlea of the astragalus, which fits into the mortise as a tenon. This trochlear surface is one fourth wider in front than behind, conforming to the divergent direction of the internal surfaces of the

malleoli. The inferior articular surface of the tibia may be described as the roof or ceiling (*plafond*) of the joint; and the articular surfaces of the malleoli have long been known as the "cheeks" of the mortise.[38] That portion of the fibula *which projects beyond the tibial plafond* is properly called the external malleolus; and the corresponding projection of the tibia is called the internal malleolus. The posterior lip of the tibial plafond projects so low as to have been called by Destot the posterior malleolus. This serves to reinforce the mortise posteriorly. It is further deepened by the transverse tibiofibular ligament which

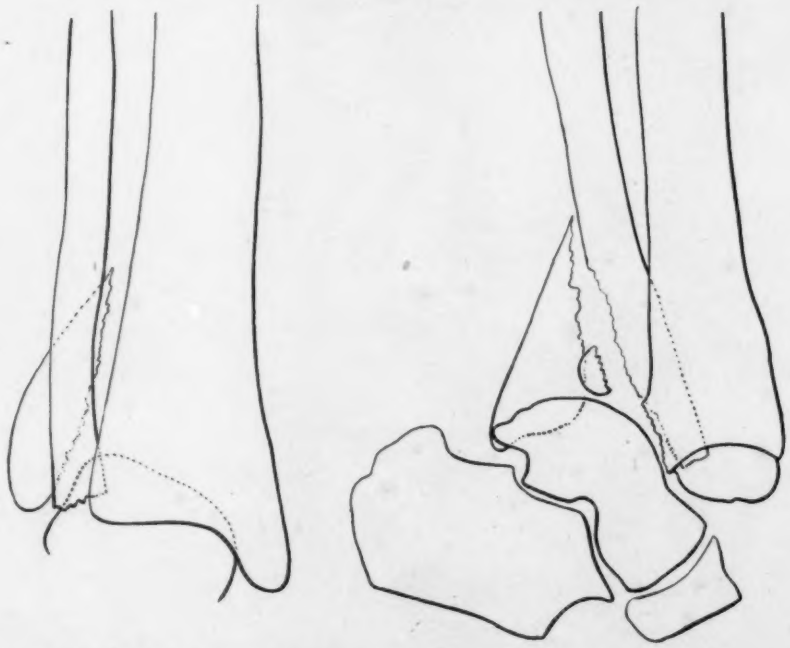

Fig. 32.—Small posterior marginal fragment in a case of fracture by external rotation. Notice the mixed oblique fracture of fibula; the intact internal malleolus (rupture of internal lateral ligament), and the complete posterior dislocation of the foot, the astragalus and the posterior marginal fragment accompanying the external malleolus.

extends from the external malleolus to the posterior lip of the tibia. Thus in walking, as the foot meets the ground in plantar flexion, the leg bones are checked in their tendency to slide forward on the astragalus by the wedge shape of the trochlea of the latter bone (broad anteriorly

38. From the Greek word for cheek, Quénu derives the adjective *génienne*, which he constantly employs in his classification and nomenclature of ankle fractures, inventing such terms as *bi-malleolaire géni-sus-genienne, géni-peronière* and *géni-supramalleolaires.*

and narrow posteriorly), by the corresponding divergence of the anteroposterior planes of the malleoli, and by the long posterior lip of the tibial plafond. In walking backward (always digitigrade, not plantigrade) the same mechanism is effective.

The fibula is attached firmly to the tibia, but a slight range of motion is permitted. The interosseous membrane extends throughout the length of the tibial and fibular shafts, the fibers running downward and laterally from tibia to fibula (as in the forearm from radius to ulna); and in addition, there are strong ligaments whose fibers run in the same direction, binding both ends of the fibula to the tibia, at which

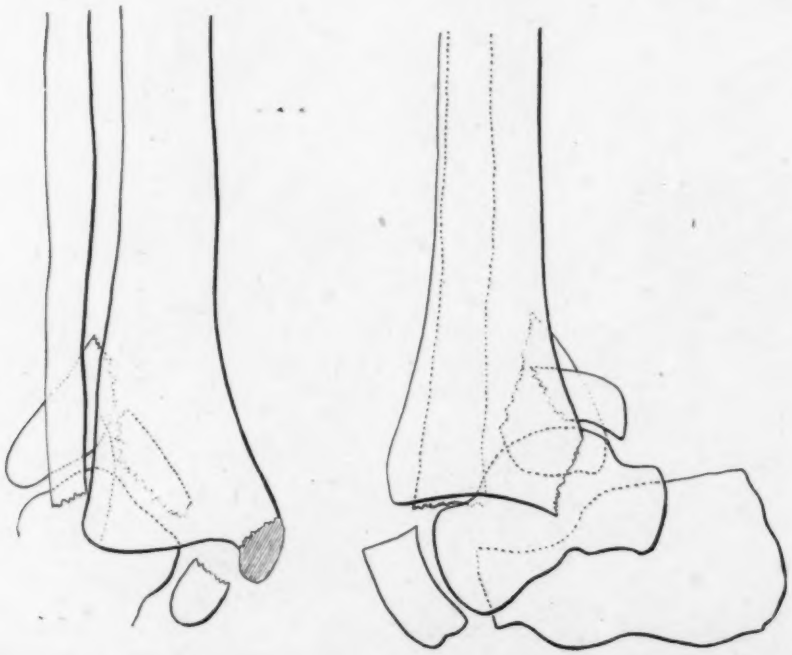

Fig. 33.—Medium-sized posterior marginal fragment in association with mixed oblique fracture of fibula and fracture of internal malleolus. The posterior marginal fragment, which is visible also in the anteroposterior view, accompanies the external malleolus and the astragalus in the very marked lateral displacement and in the incomplete posterior dislocation.

points only are the two bones in contact. At the upper end, where the head of the fibula butts against the overhanging external condyle of the tibia, there is a synovial cavity to the tibiofibular joint; but at the lower end, where the fibula is received into a longitudinal groove between the anterior and posterior tubercles on the lateral surface of the tibia (Fig. 4), no such joint cavity exists, union being effected by a dense feltlike interosseous ligament, reinforced anteriorly by the anterior

inferior tibiofibular ligament, and posteriorly by the posterior ligament of the same name. The malleoli, of which the external is the longer and is situated more posteriorly, serve to keep the foot (which is appended to the astragalus) under the leg bones. The astragalus itself has no muscles attached to it, and serves only as a ball in a ball-bearing joint to facilitate movements of the leg bones above it and of the tarsal bones below and in front. The foot is attached to the leg bones by

Fig. 34.—Large posterior marginal fragment in association with mixed oblique fracture of fibula and fracture of internal malleolus; only moderate displacement, chiefly due to the outward rotation.

ligaments, of which the lateral portions are best developed, constituting for the ankle, as in other hinge joints, lateral ligaments which hinder motion except in the anteroposterior plane. The internal lateral ligament passes from the internal malleolus in radiating direction (1) anteriorly to the scaphoid and median surface of the calcaneum (sus-

tentaculum tali) and (2) posteriorly to the median tubercle on the posterior surface of the astragalus. The external lateral ligament has three distinct bands, passing from the external malleolus: one goes forward to the lateral border of the neck of the astragalus, just above the sinus of the tarsus; the middle band passes downward and slightly backward to the calcaneum; while the posterior, whose deep portion is extremely strong (Fig. 5), is attached to the lateral tubercle on the posterior surface of the astragalus (os trigonum), which, being from 5 to 7 mm. posterior to the median tubercle, is the portion of the

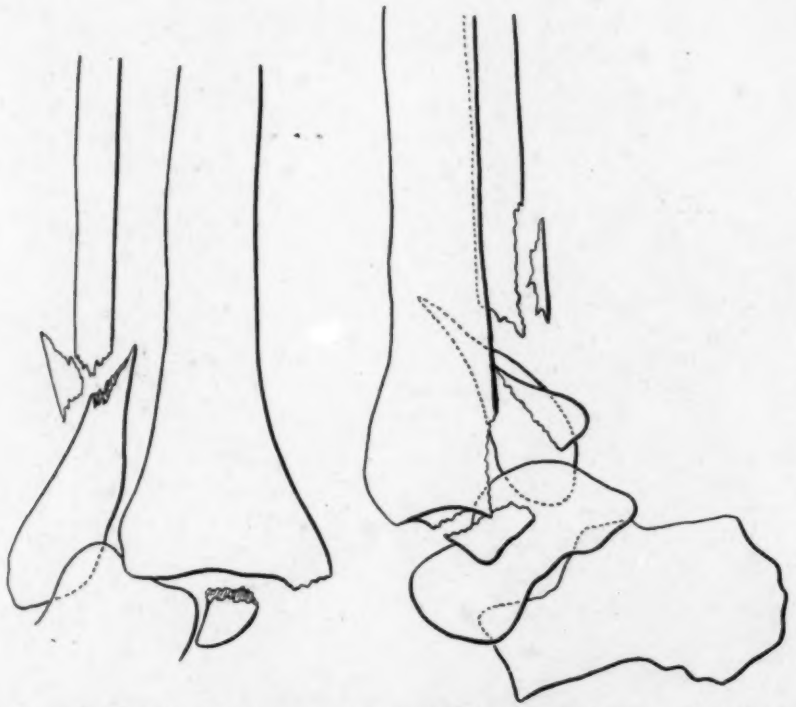

Fig. 35.—Small posterior marginal fragment in association with abduction fracture of fibula and fracture of internal malleolus. Note the diastasis, the typical wedge detached from the flexion surface of fibula and the posterior marginal fragment accompanying the external malleolus and astragalus in their marked lateral and posterior displacement.

astragalus which casts the farthest posterior shadow in lateral roentgenograms of the foot. This posterior band of the external lateral ligament is so exceedingly strong that it is very seldom ruptured; it holds the astragalus almost indissolubly attached to the external malleolus,[39] and

39. Chaput (Bull. et mém. Soc. de chir. de Paris **38**:1192, 1912.) says they are like Siamese twins.

in injuries of the ankle either one or other bone to which the ligament is attached is more easily broken than is the ligament ruptured. As will be shown subsequently, usually the fibula gives way when the strain comes; occasionally, however, the posterior tubercle of the astragalus is detached; and I have seen at least one case in which fracture occurred at both points simultaneously.[40]

The next question that arises is, what is the function of the fibula?

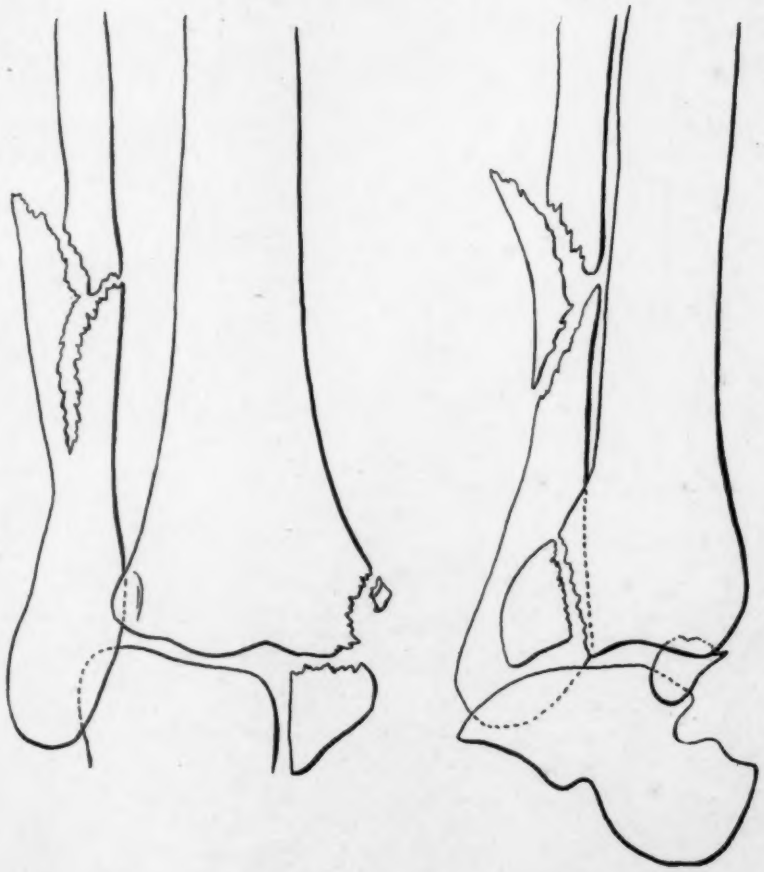

Fig. 36.—Posterior marginal fragment associated with diastasis and fracture of internal malleolus, and with fracture of fibula by bending backward (flexion surface posterior, extension surface anterior) as evidenced by wedge detached from posterior surface of fibula.

40. The posterior tubercle of the astragalus may sometimes be fractured by direct violence as it is crushed against the posterior lip of the tibia in forced plantar flexion of the foot.

Humphry[41] (1858) notes that the fibula is an inconstant bone in animals: in carnivora and pachydermata it extends from the upper end of the tibia to the ankle as in man. In most rodents it is united with the tibia at the lower part. In ruminants it altogether disappears. In birds its upper extremity enters into the knee joint and articulates with the external condyle of the femur; it lies close against the tibia and dwindles and disappears about the middle of the leg. In reptiles, it is large, in many extending to the knee joint above and

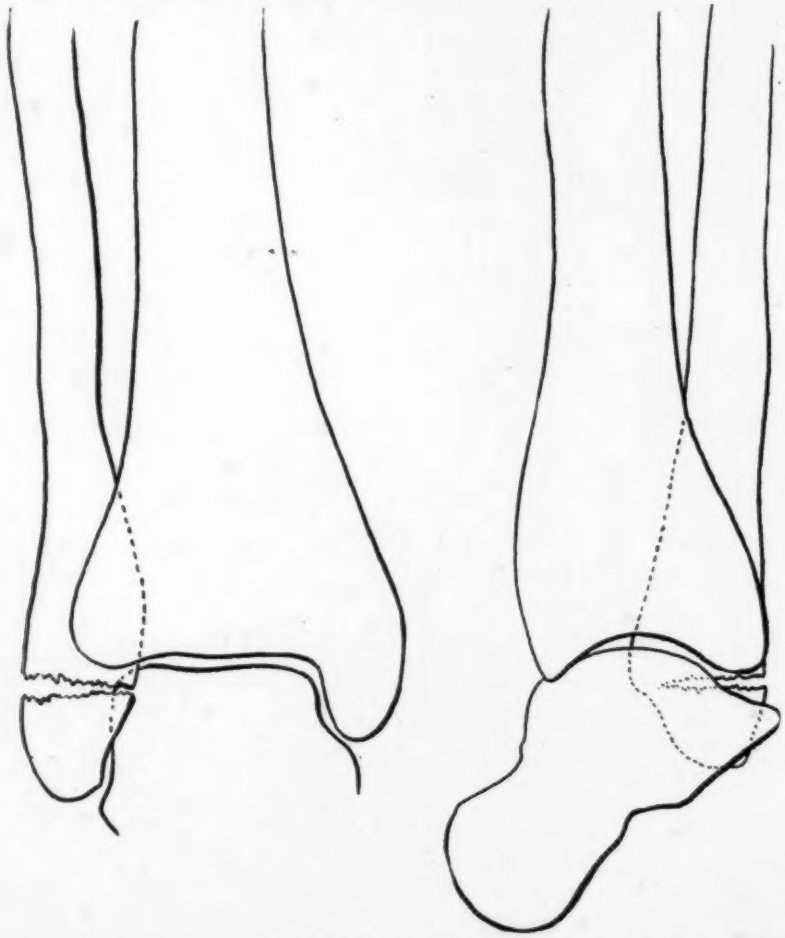

Fig. 37.—Fracture by adduction, first degree (C, I); avulsion of external malleolus. See page 118.

to the tarsus below. In the bat, the lower half of the fibula and the upper half of the ulna are retained.

It has seemed to me, from study of the skeletons in the Museum of the Academy of Natural Sciences of Philadelphia, that the fibula was best developed and extended farthest beyond the tibia at the ankle in those animals which

41. Humphry: Treatise on the Human Skeleton, Cambridge, 1858, p. 490.

were most nearly plantigrade, and in which, as in man, stability rather than agility was demanded. In digitigrades, such as the horse and camel, there is no fibula; in partial or less complete digitigrades (rhinoceros) it extends beyond the tibial plafond, but not below the level of the internal malleolus. In a still less complete digitigrade (almost a plantigrade), such as the elephant, it extends below the level of the internal malleolus, and there is outward rotation of the lower end of the tibia, as in man. In the gorilla, chimpanzee, orang-utan, etc., on the other hand, which are more plantigrade than digitigrade (but in which, as already noted, agility is retained at the expense of stability),

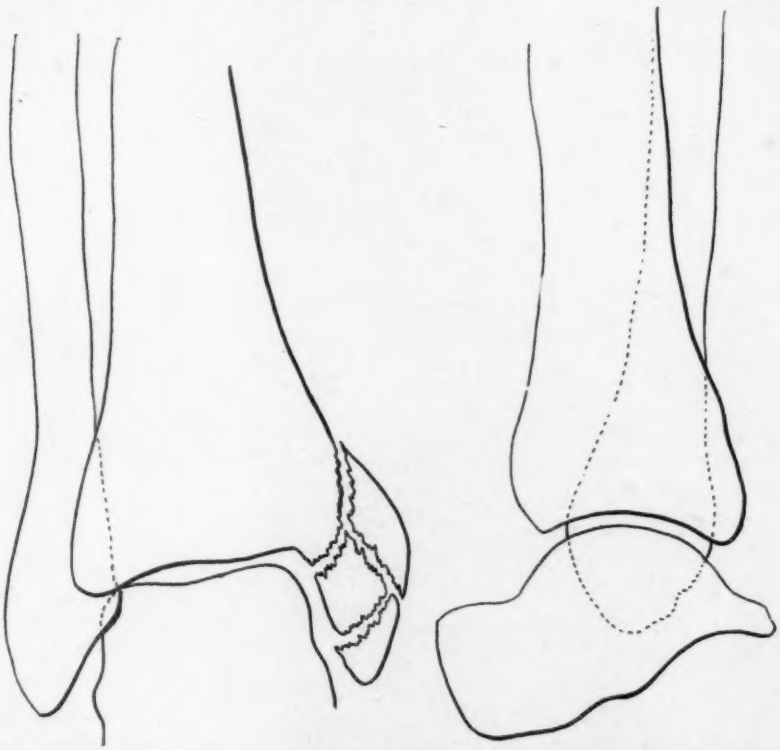

Fig. 38.—Adduction fracture, second degree (C, II, a); avulsion of external malleolus, represented by partial rupture of anterior fibers of external lateral ligament (rare) followed by compression fracture (note the comminution) of internal malleolus.

the lower end of the tibia has not rotated out as far as in man (in fact, not so far out as a transverse plane through the tibial condyles), and the fibula descends no lower than the internal malleolus. This lack of development of the fibula in these more or less anthropoid animals was noted by Bland-Sutton[42] (1888); and he further calls attention to the fact that in babies born with congenital clubfeet this deformity is merely a lack of normal development, the outward rotation of the lower end of the tibia not having occurred, and the external malleolus not having descended below the level of the internal.

42. Bland-Sutton: Am. J. M. Sc. **95**:376, 1888.

Evidently, he concluded, the external malleolus was developed only to aid members of the human race to walk in the erect posture; it was required to keep the foot steady and prevent it from turning outward into a position of extreme valgus. As Destot says, the external malleolus acts merely as a splint to maintain the direction of the foot.

But if this is so, why is the fibula a separate bone in man? Why is not the external malleolus merely a part of the tibia? To this I know of no better answer than that given by Bromfeild[11] (1773) that if it were a part of the tibia,

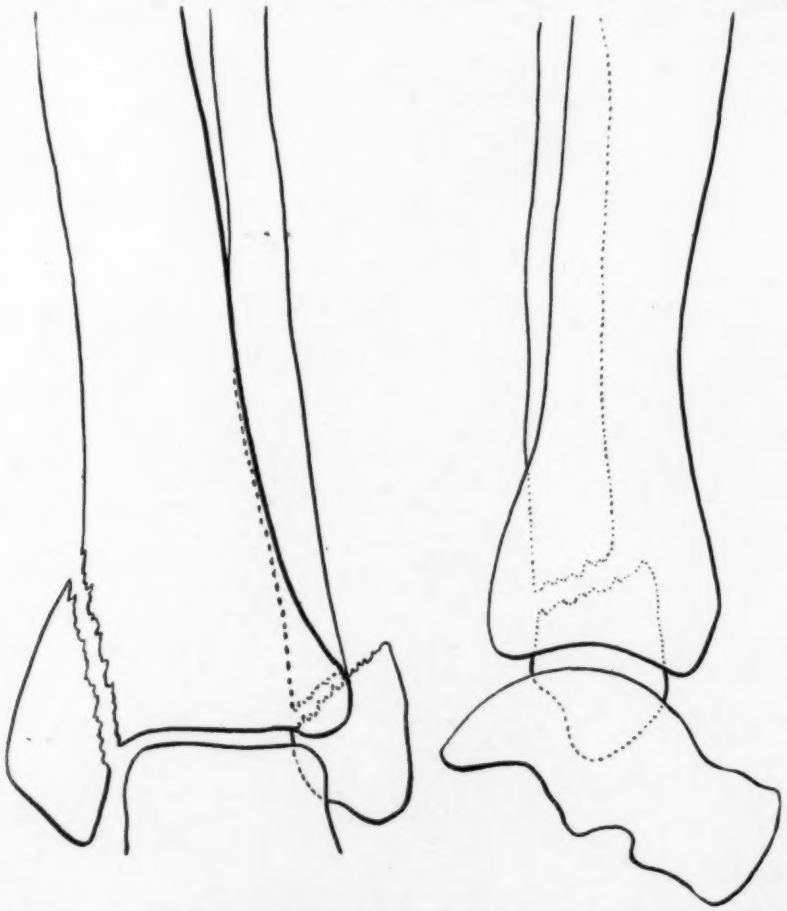

Fig. 39.—Adduction fracture, second degree (C, II, a); avulsion of external malleolus followed by compression fracture of internal malleolus.

and no give or play whatever occurred between the malleoli, no one could take more than a very few steps without fracturing one or other malleolus.

If a leg is studied from which all soft parts have been removed except the ligaments and the interosseous membrane, these phenomena may be observed:

In full plantar flexion (extension) of the foot, the anterior and middle bands of the external lateral ligament become tense and pull the external malleolus medially (and slightly downward and backward) against the tibia, keeping

the external malleolus in close contact with the astragalus as this glides forward and presents to the intermalleolar space a slightly less diameter than in full dorsiflexion of the foot. In the latter movement, dorsiflexion, the anterior band of the external lateral ligament becomes lax, and as the external malleolus is forced away from the tibia and slightly upward, the anterior and posterior tibiofibular ligaments become tense, especially the anterior ligament; also, as flexion beyond a right angle occurs, much tension develops on the middle band of the external lateral ligament, and this pulls the malleolus backward: the posterior band of the external lateral ligament is always tense; it makes the astragalus and external malleolus practically one bone.

Fig. 40.—Adduction fracture, second degree (C, II, b); large tibial fragment extending into shaft replaces compression fracture of internal malleolus.

The expansion of the intermalleolar space which occurs during dorsiflexion of the foot may amount to several millimeters. I have measured it by affixing a wire in each malleolus and bending the ends of these wires forward over the ankle joint until they crossed each other in parallel lines: by scratching a mark on each wire at the same point when the foot is in full plantar flexion, it is easy to measure the excursion as the foot is brought up into full dorsiflexion. This excursion, which exceeds two mm. and may approach three mm., allows the wider anterior diameter of the articular surface of the astragalus to pass back between the malleoli in dorsiflexion, while the downward drag of the

anterior and middle bands of the external lateral ligament keeps the malleoli in contact with the small posterior diameter of the astragalus which presents between them in full plantar flexion. Destot[8] (1911) pointed out that inasmuch as the intermalleolar axis and the axis of rotation of the astragalus, in flexion and extension, do not coincide (they form an angle of about 30 degrees, open laterally, Figure 4), it is not a directly transverse diameter of the astragalus that presents between the malleoli at any point of flexion or extension, but a

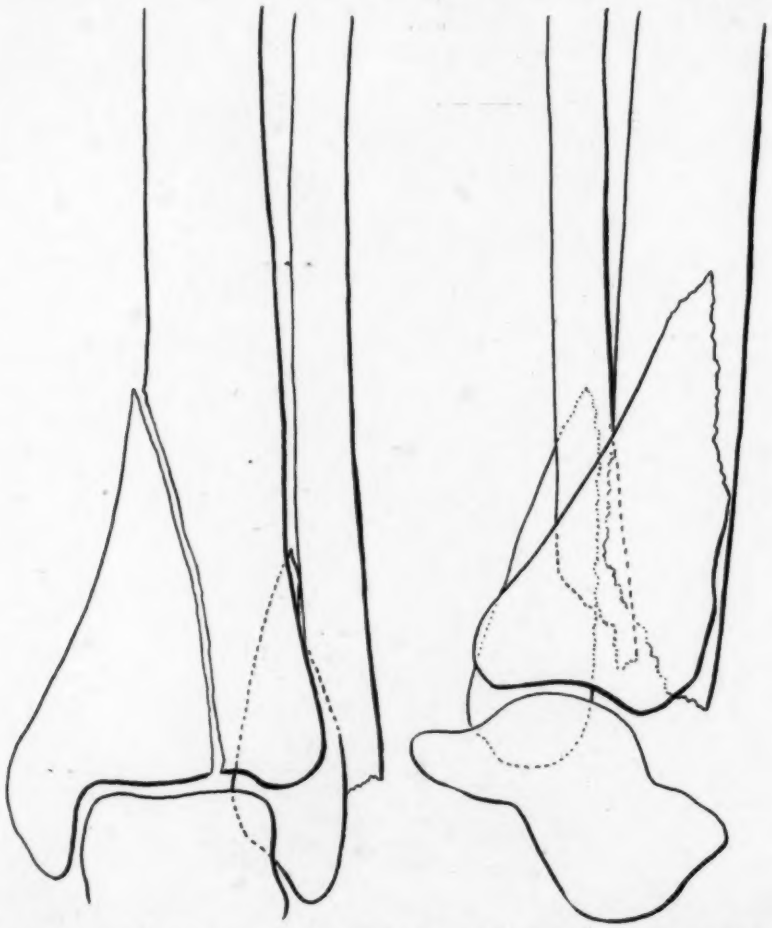

Fig. 41.—Adduction fracture second degree (C, II, b); spli.ting fracture of median articular surface of tibia replaces crush of internal malleolus. See page 118.

diameter of varying obliquity which is, however, always nearly the same in length; and he is inclined to ignore the existence of the movements of the external malleolus. And I have dwelt at some length on these movements because I have found them nowhere described and because there has been some dispute about them. Humphry[41] (loc. cit., p. 557) asserted that the increase of the distance between the malleoli was secured solely by the elasticity of the fibula, which bent inward toward the tibia in its lower fourth, when the

external malleolus was forced outward. Nancrede[43] (1880), however, pointed out that an upward and downward movement of the fibula occurred, and asserted that Humphry's theory was "preposterous and untrue."[44] This is my own opinion also.

The upper end of the fibula can also be seen to move in flexion and extension of the foot at the ankle; in full plantar flexion (with median and downward movement of the external malleolus), the superior end of the fibula moves

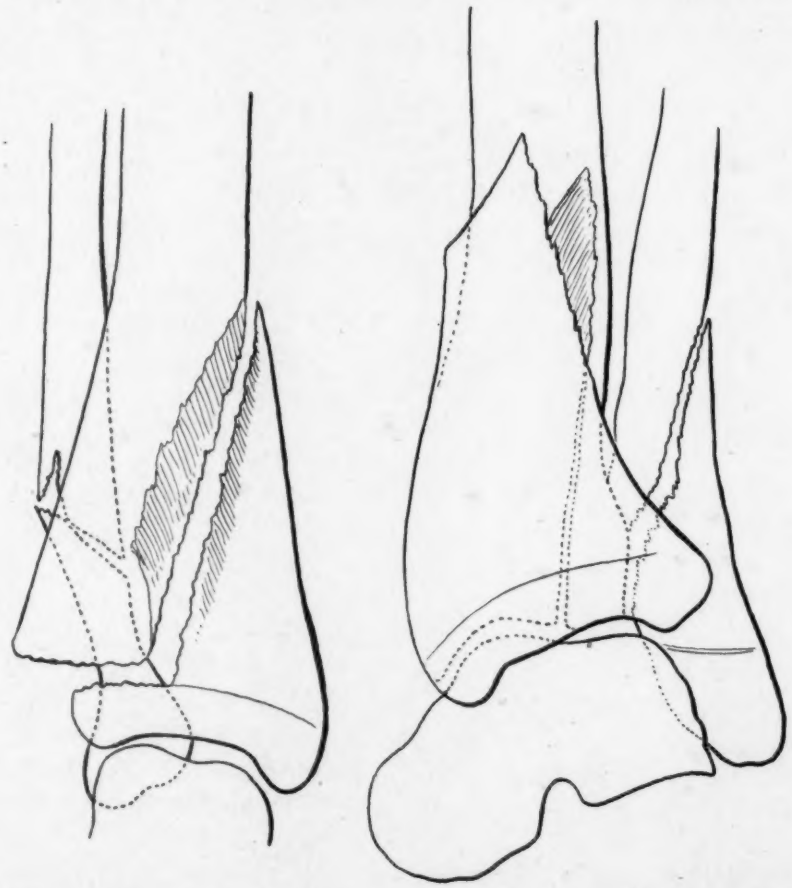

Fig. 42.—Adduction fracture, third degree (C, III); detachment of lower epiphysis with splitting of median surface of tibia.

slightly forward and rotates slightly outward, its anterior surface turning away from the tibia. This movement is due largely to the median and backward pull exerted on the external malleolus by the middle and posterior bands of the external lateral ligament. The head of the fibula slides backward and very slightly upward again in full dorsiflexion. Thus the chief movement of the

43. Nancrede: Phila. M. Times **10**:316, 1880.
44. Nancrede: Maryland M. J. **7**:76, 1880.

superior tibiofibular joint is an anteroposterior one (downward and forward, or upward and backward) around the inferior tibiofibular joint as a pivot; but these movements are so slight as to be scarcely appreciable.

Very little change occurs in the interosseous membrane during these movements, except in its lower fourth, where it spreads and becomes tense (the aperture for the anterior peroneal artery tends to become round from oval) as the external malleolus ascends and moves backward; and it again becomes relaxed when the external malleolus descends and moves mesially toward the

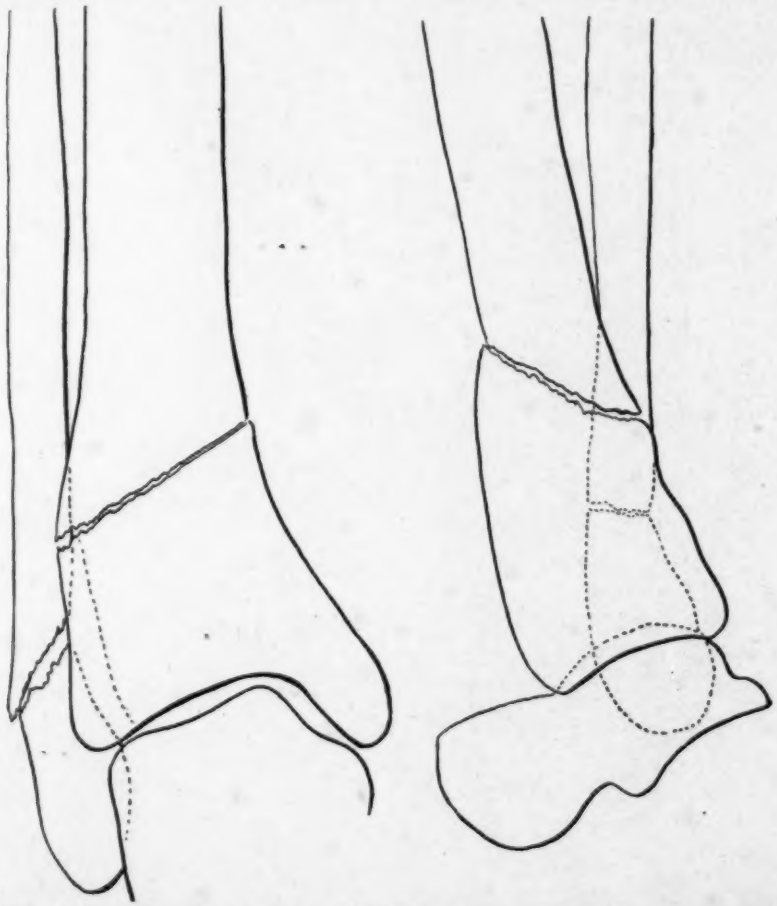

Fig. 43.—Adduction fracture, third degree (C, III); the supramalleolar fracture by adduction (type produced experimentally by Tillaux). See pages 71 and 119.

tibia. The strength of the interosseous membrane is much greater than usually supposed. Even when the lower end of the fibula is freed from its tibial attachments, very great force is required to rupture the interosseous membrane, and fracture of the fibula in its lower third is the nearly invariable sequel.

The normal movements of the ankle joint are those of flexion and extension—20 degrees of dorsiflexion and 60 degrees extension or

plantar flexion, a total of 80 degrees approximately. This motion occurs around an axis which passes in the frontal plane somewhat below and in front of the tip of the external malleolus. This axis makes an angle of 30 degrees (thereabouts) with the bimalleolar axis. This arrangement accounts for the greater excursion forward of the lateral astragalar surface in relation to the external malleolus as compared with the motion which occurs between the median surface of the astragalus and the internal malleolus; as well as for the apparent deviation of the point of the foot medially in full plantar flexion, and laterally in full dorsiflexion.

If movements were possible in the ankle joint around an antero-posterior axis, they should be named adduction (tibial flexion) and abduction (fibular flexion), or movements in the frontal plane toward and away from the median line. These movements are quickly resisted by the tension on the lateral ligaments (Figs. 6 and 7), and if forced, the malleolus *away from which motion occurs* is torn off by its ligament, or the ligament itself ruptures (see the experiments of Hönigschmied, related on page 72). Motions of adduction and abduction in the foot normally occur in the subastragalar joint and permit the calcaneum without difficulty to accommodate itself to slight irregularities of the soil. But the calcaneum is attached to the astragalus by the extremely strong astragalocalcanean interosseous ligament; and when such movements are too extensive, they are transmitted directly to the astragalus and from it to the tibiofibular mortise where fracture of one or the other malleolus is the usual consequence. Not until that malleolus on which the pull comes has given way, or the corresponding ligament has ruptured, is the astragalus able to act on the other malleolus by a push so as to produce a compression fracture. Rare exceptions to this general rule occur, however, as when the calcaneum is itself first broken, and becomes so displaced as to press directly on the end of the fibula, producing a compression fracture of the external malleolus (Fig. 23).

Movements of rotation around the long axis of the leg may be attempted in the ankle joint by twisting the point of the foot toward or away from the median line. As pointed out by Maisonneuve[4] (1840), movements of inward rotation are almost inseparable from a movement of adduction, as the numerous joints in the anterior tarsus render the foot very mobile in this direction. Any movement toward outward rotation, however, converts the foot into a rigid lever, and motion is easily and with much force transmitted to the ankle joint, the astragalus attempting to turn so as to bring its long axis crosswise between the malleoli. Owing to physical laws, it is on the external malleolus that the greatest strain comes. Maisonneuve illustrated this by placing a ruler (which represents the foot) between two parallel

volumes (which represent the malleoli): the volume which is moved is always that toward which the long end of the lever moves (Fig. 8); even if this volume be much heavier than the other it is easily moved by the greater leverage exerted. In movements of outward rotation, the foot, relatively to the tibia, is a lever of the first order, with its fulcrum on the anterior border of the fibula: the arm of the resistance will have, say, a length of 3 cm., that of the power 12 cm. (the length of the foot being taken as 15 cm. from the toes to the posterior border of the ankle joint). Relatively to the fibula, it is a

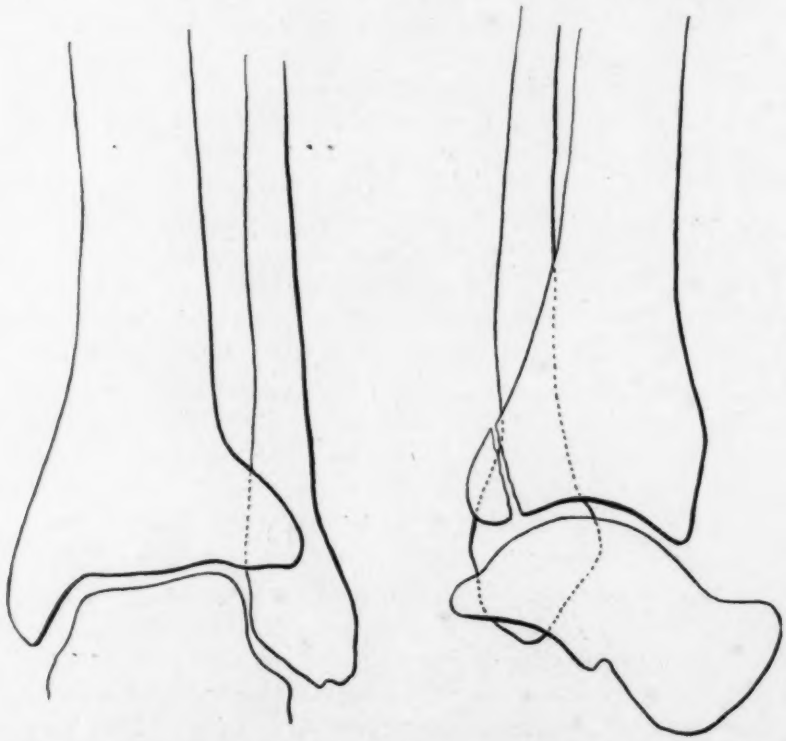

Fig. 44.—Isolated fracture of posterior tibial margin; no displacement; from compression upward and backward.

lever of the second order, with its fulcrum at the posterior border of the internal malleolus: the resistance, therefore, has an arm of 3 cm. (as in the other case) but the power has an arm of 15 cm. (the whole length from the point of the foot to the fulcrum). Thus the force which tends to fracture the fibula is as 12 is to 9, or as 4 is to 3. Such a mechanism as this (outward rotation of the foot) usually causes an oblique fracture of the lower end of the fibula (see Hönigschmied's [6] experiments, page 72); and if such a fracture be made

by an osteotome it will be found that *external rotation is the only movement that will cause separation of the fragments* (Fig. 9). It is evident that the mechanism of this fracture, which is the most frequent of all fractures of the ankle (more than 25 per cent. of all cases), involves not only a push outward on the anterior border of the external malleolus, as noted by Maisonneuve, but also, as Hönigschmied pointed out, a pull inward on its posterior border by means of the posterior band of the external lateral ligament. The line of this fracture is oblique from above and behind, downward and forward. It is, properly speaking, a spiral fracture produced by torsion. Its obliquity varies greatly; but it is always higher on the posterior surface of the fibula than on its anterior, and the line of fracture passes through and involves the inferior tibiofibular joint. Almost invariably, its lower and anterior end extends to the external malleolus (in 90 per cent. of our cases): often just below the tibial plafond, sometimes as far down as the very tip of the malleolus. Thus in practically every instance the anterior inferior tibiofibular ligament remains intact, or even if partially ruptured, there results no true diastasis between fibula and tibia. At most, the lower fragment, comprising that part of the fibula posterior to the attachment of the anterior tibiofibular ligament, rolls outward and slightly backward around the unruptured posterior tibiofibular ligament as a hinge.

If this "mixed oblique" [45] fracture of the fibula, as Destot names it, is the sole lesion resulting from outward rotation of the foot, there is little or no displacement (Figs. 10 and 11). This was the case in all of the seventy-nine cases studied by Dr. Bromer and myself. If the force continues to act, the next lesion which is added is rupture of the internal lateral ligament (in twelve cases only, in our series), or, far more frequently, fracture of the internal malleolus, usually only of its anterior tip, seldom of its whole extent (Fig. 12). This combined lesion (oblique fracture of the fibula with fracture of the internal malleolus) occurred in thirty-two cases in our series, or in 10 per cent. of the entire number.[46] The displacement may be slight or marked. And in very many cases (fifty-one additional cases in our series), besides these two lesions, there is added the complication of fracture of the posterior margin of the tibia. Counting in all complications and variations, this type of fracture occurred in 100 cases or in 33 per cent. of the total 300 cases we have studied.

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45. "Mixed" because involving the fibula both above and below the tibiofibular joint.

46. Chaput (Bull. et mém. Soc. de chir. de Paris **32**:1047, 1906) noted that among 130 cases of fracture at the ankle, of which he had studied the roentgenograms, 113 conformed to this oblique type of fracture of the fibula, against only seventeen cases in which the fracture was clearly above the malleolus.

Seldom does the obliquity of the fibular fracture pass so high as to be above the level of the anterior inferior tubercle of the tibia (Figs. 13 and 14): in three cases this tubercle was detached (in two of these there was no appreciable lesion at the internal malleolus, but in one, the internal malleolus was fractured), and in two others it is probable that a disjunction of the joint had occurred, as indicated by suggestive roent-

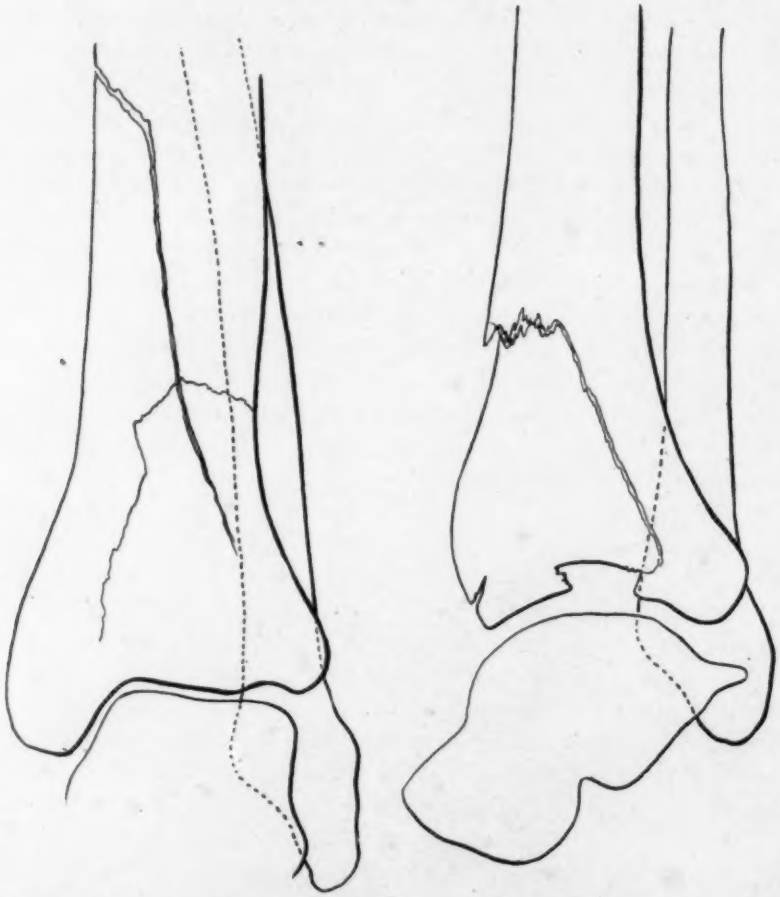

Fig. 45.—Comminuted fracture of tibial plafond; from compression upward.

gen-ray findings. This lesion (fracture of the anterior tibial tubercle, or diastasis of the tibiofibular joint) must occur previous to, even if nearly simultaneously with, the oblique fracture of the fibula;[47] because

47. Lapointe (Souligoux: Bull. et mém. Soc. de chir. de Paris **44**:1042, 1914) quite justly contends that if fracture of the anterior tubercle can occur as an isolated lesion (as in the case he reported), it must be admitted it may also occur as the first lesion of a more complicated fracture.

after the fibula is fractured its lower fragment (on which alone the force is acting) is already detached from the anterior tubercle of the tibia, and a continuance of the force would merely increase the separation. Moreover, there is lack of displacement in these isolated oblique fractures of the fibula merely because to permit displacement it is necessary that the internal malleolus (or its ligament) previously give way. If a tibiofibular diastasis (with or without separation of the tubercle) occurs (and if it occurs it must always occur previous to a fracture of the fibula, as already remarked), then the fracture of the fibula (by torsion still) occurs not through the inferior tibiofibular joint but above it, sometimes through its surgical neck, often through its true neck in the upper third of the fibula, as originally pointed out by Maisonneuve. That such a lesion in the upper third of the fibula can occur without appreciable bony lesion at the ankle cannot be denied. We have two such cases; and Quénu [48] went so far as to say that many more such fractures occur without diastasis than with it; and that they may occur even without any lesion at all at the ankle joint (Fig. 15).[49] It must also be recognized that diastasis is not necessarily followed by fracture of the fibula at any level, as shown in the lesion represented in Figures 16 and 17 in which the fibula detached the anterior inferior tubercle of the tibia, and in which, after rupture of the internal lateral ligament had occurred, the fibula was forced by the astragalus around back of the tibia by continuance of external rotation of the foot, as in the mechanism described by Huguier [50] (1848), though in his cadaveric experiments, as well as in the case illustrated by Destot (Fig. 67, p. 142 of his monograph), this displacement was accompanied by (and I believe succeeded by) a fracture of the upper end of the fibula.

In rare instances, the avulsing force on the internal malleolus may be so great as to cause fracture of the entire lower end of the tibia (or in children a separation of the epiphysis [51]): this appears to have been the mechanism in four of our cases, which on a purely anatomic classification should perhaps be grouped with the supramalleolar fractures (Figs. 18 and 19).

The significance of the posterior marginal fragment, and the mechanism by which it probably is produced, will be discussed in another place (p. 112).

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48. Quénu: Rev. de chir. **35**:897, 1907.

49. Long recognized as possibly due to pull of the biceps muscle. See a study by Lonhard (Deutsche mil.-ärztl. Ztschr. **43**:219, 1914).

50. Huguier: Union méd., Paris **2**:120, 1848.

51. In addition to the 300 cases of fracture of the ankle listed on page 122, we have records of at least nine cases diagnosed as epiphyseal separations; but as in these the roentgenograms available showed no gross separation at the epiphyseal line of the tibia, we have not included them in our statistics.

I desire to return now to the movement of *forced abduction,* a discussion of which has been intentionally postponed until disposition had been made of the much more frequent mechanism (outward rotation). We find in any large series of fractures at the ankle a certain number in which fracture of the internal malleolus is the only lesion (in our series this lesion occurred in more than 6.5 per cent. of the whole number). Now it is not rational to suppose that the same mechanism which at one time causes an isolated oblique fracture of the lower end of the fibula will at another cause an isolated fracture of the internal malleolus: they must be produced by different mechanisms. Experi-

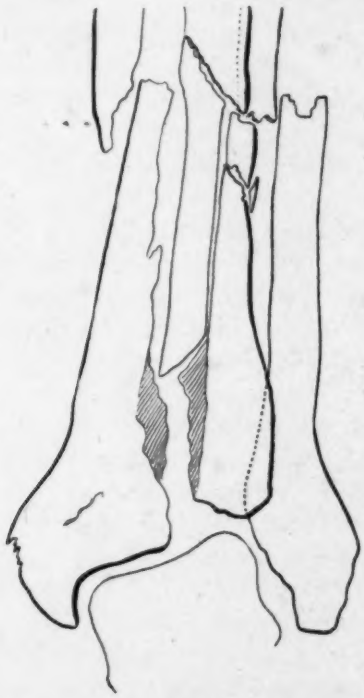

Fig. 46.—T or Y fracture involving ankle joint; from compression upward.

mentally, it is very clearly seen (note the experiments of Hönigschmied, detailed on page 72, that straight abduction (fibular flexion) of the foot has as its primary and most constant lesion fracture of the internal malleolus, or its equivalent, rupture of the internal lateral ligament. This is a prerequisite in order to free the astragalus sufficiently so that it may press directly on the external malleolus, which it does by rotation around an anteroposterior axis. The experiments of Bonnet [52] (1845), have been overlooked by most students. He showed, long

52. Bonnet: Traité des maladies des articulations, Lyon **2**:428, 1845.

before Tillaux or Hönigschmied, that abduction (fibular flexion) of the leg, while the foot was held in a vise, caused: first, fracture of the internal malleolus or rupture of the internal lateral ligament (Fig. 20), and, if the abduction was increased, a crushing or fracture of the external malleolus (Fig. 21), never of the fibula above the inferior tibiofibular joint. But sometimes no lesion of the external malleolus was caused even when the internal malleolus was widely separated. If, on the other hand, abduction of the foot was produced with the leg lying on its fibular side, but with the foot projecting free of the table, the same lesions occurred at the internal malleolus; but the fibula broke above the inferior tibiofibular ligaments at the point where it rested on the table.

In the mechanism of these fractures by abduction the influence of the tibiofibular ligaments is paramount:

1. If the tibiofibular ligaments hold, the fibula breaks across through the external malleolus proper (i.e., below the tibiofibular ligaments) and not above these ligaments by that "preposterous and untrue" mechanism to which Nancrede objected, namely, the inward bending of the fibula toward the tibia. It is not proper, perhaps, to deny that the latter mechanism might sometimes occur (all things are possible) in a patient with exceedingly relaxed ligaments; but I feel strongly inclined to state in the words of Souligoux (applied by him to the existence of an isolated fracture of the posterior tibial margin) that I do not believe such a mechanism exists, and will not believe it exists until somebody shows me its method of production. Of the 300 fractures which we have studied, we find only thirteen cases which seem to belong to this type (bimalleolar fracture by abduction): evidently the more nearly the movement of the foot conforms to the type of straight abduction, the more apt is diastasis, and as a consequence, fracture above the tibiofibular ligaments to occur (see below); while the more nearly it corresponds to external rotation (deviation of the point of the foot outward), the more certain is the fracture to be oblique in type, involving the inferior tibiofibular joint, but, as already explained, causing no true diastasis; hence the extreme rarity of true bimalleolar fractures by abduction (Fig. 22).[53]

2. If the tibiofibular ligaments rupture, then the fibula is freed from the tibia, and if the force continues (in life it is now the weight of the body borne chiefly, or at least abnormally, on the fibula), the fibula breaks "by flexion" and the break usually occurs where the fibula is weakest, through the surgical neck above the inferior tibiofibular ligaments. Study of roentgenograms or museum specimens of fractures

53. Mention has already been made (p. 99) of isolated fracture of the external malleolus associated with fracture of the calcaneum (Fig. 23).

of this type shows clearly two things: an evident tibiofibular diastasis, and a flexion fracture (Biegungsbruch) of the fibula. The typical mechanism of the flexion fracture is illustrated in Figure 24 (p. 79), copied from Messerer; and though at first glance some roentgenograms of fractures of the fibula at this height may not seem to indicate this mechanism clearly, more careful study (in nearly every case, at least, with which I am familiar) shows that the fracture conforms to the Biegungsbruch type: thus the line of fracture is either nearly transverse (rare), slightly oblique (frequent, Fig. 25), or (very frequent) is comminuted in the typical manner, with detachment of a wedge-

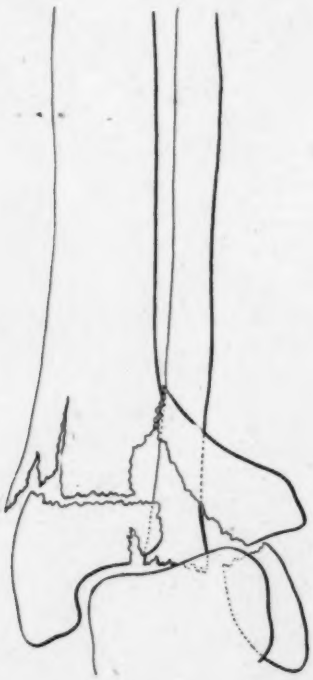

Fig. 47.—T or Y fracture involving ankle joint; from compression upward.

shaped fragment from the concavity of the bent bone (Figs. 26 and 35); that is to say, the wedge is on the lateral border of the fibula (apex toward the tibia) if the fibula was broken by straight abduction of its lower end, or on the posterior border (apex anteriorly) if it broke by posterior displacement of its lower end (Figs. 34 and 36).

Now it is a prerequisite for a bone (or any other similarly shaped structure) to be broken by indirect force through bending that one of its ends must be fixed and the other end movable. The upper end of the fibula is fixed by its attachments at the superior tibiofibular joint

and by the interosseous membrane; and to permit fracture by bending of its shaft, under such circumstances, by means of indirect force applied to the external malleolus, it is first of all necessary that the lower end of the fibula be freed from its attachments to the tibia. If these attachments are not freed, it is extremely unlikely that a fracture by bending can occur; though it is possible to conceive of an exceptional instance in which such an event might occur, as, for instance, if the surgical neck of the fibula (as in the second mechanism described by Bonnet) were pressed against the edge of a table or similar object by abduction of the foot; but in such a case the action of direct violence in causing the fracture could not be excluded. In life it would be less unlikely for a fracture by compression to occur (the force being transmitted in the long axis of the fibula which was still rigidly attached to the unbroken tibial shaft); whereupon the fracture would present a very different appearance (Fig. 15); or even less unlikely for a fracture by torsion to occur, though for this mechanism also it is necessary for the two ends of a bone to be movable in opposite directions or for one to be fixed and the other movable. Torsion was held by Maisonneuve (and no doubt correctly) to be the mechanism by which was produced the fracture in the upper third of the fibula described by him as "fracture par diastasis;" and it is this mechanism (torsion) which causes it to be situated so close to the fixed end of the bone,[54] and which makes me class it as a variant of the fracture "by outward rotation of the foot" already discussed; and which convinces me that it does not belong in the same class with fractures produced by straight abduction of the foot, in which cases, I repeat, the fracture of the fibula occurs in its lower third and is caused by a bending mechanism.

It is true, of course, that the force may cease to act before the fibula breaks; in which case merely a diastasis results (Fig. 27).

Careful study of thirty cases of fracture of the fibula through its surgical neck in our series has shown the characteristics of a fracture by bending in all except two cases, in which it was clearly by torsion. When this lesion (by whatever mechanism) was unaccompanied by a diastasis of the inferior tibiofibular joint (or its equivalent, detachment of the anterior tubercle of the tibia), then the history has shown (in all but one case in which the history is unknown) either that the fracture was due to direct violence (Fig. 28) or that the clinical signs of a tibiofibular sprain were present though no diastasis was shown by the roentgen ray. Quénu[55] (1912), however, for whose opinion every student of fractures has great respect, held that diastasis was not a neces-

54. Once the lower end of the fibula is freed from the tibia, the interosseous membrane presents no obstacle to torsion.

55. Quénu: Rev. de chir. **45**:242, 1912.

sary accompaniment of this type of fracture, though he acknowledged its extreme frequency, a fact to which he had called attention in 1909; and as early as 1907, he had pointed out that this type of fracture was that which was most frequently accompanied by diastasis. But he was at that time (1907) inclined to the view that the fracture of the fibula occurred simultaneously with or even before the diastasis. But Destot [56]

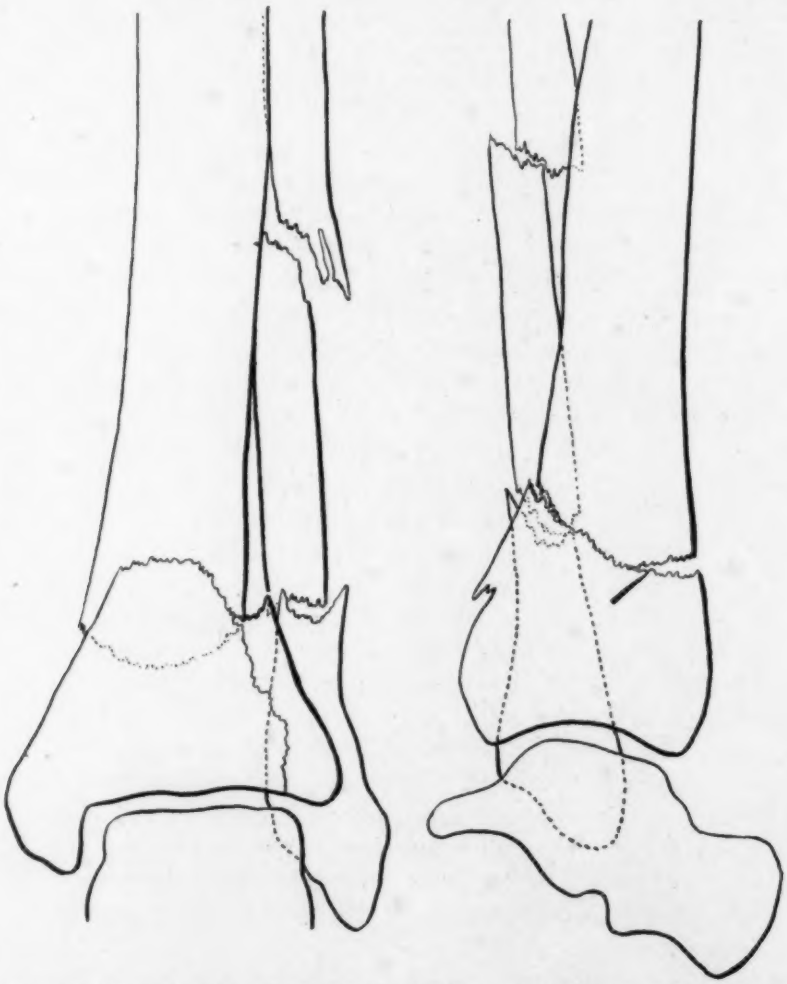

Fig. 48.—Comminuted supramalleolar fracture; probably by direct violence.

(1912) is firm in his belief that fracture of the fibula above the inferior tibiofibular joint demands as a preliminary a sprain or a diastasis of that joint. Is not then the legend to Figure 67, p. 142, in his monograph

56. Destot: Lyon chir. **8**:245, 1912.

(1911) inaccurate when it states that the diastasis shown was possible only because of the existence of a fracture near the head of the fibula?

Finally, as in fractures by outward rotation, the most advanced stage of abduction fractures may be regarded as one in which the entire lower end of the tibia is torn off as the representative of the internal malleolus; but in these also the fibula breaks characteristically by flexion through its surgical neck (Fig. 29)—it is not a mixed oblique fracture as in the third degree of fracture by external rotation already described (Figs. 18 and 19).

Fig. 49.—Anteroposterior view of ankle joint. See page 124.

Diastasis and Lateral Displacements of the Foot.—It is well to analyze more carefully what is meant by diastasis of the inferior tibiofibular joint.[57] It is easily seen in the prepared specimen that in all movements of the foot there is greater strain on the anterior than on the posterior inferior tibiofibular ligament; and that if the anterior

57. The criteria of diastasis from a roentgenographic study will be discussed by Dr. Bromer in his appendix to this paper.

ligament alone is divided, a separation of the fibula from the tibia to the extent of about 1 cm. becomes possible anteriorly, the fibula still being attached by the interosseous ligament and the posterior tibiofibular ligament. This degree of separation is sufficient to constitute a diastasis; lesser degrees, with incomplete rupture of the anterior tibiofibular ligament, and therefore without separation, constitute a sprain. But in many cases in which a temporary diastasis (disjunction) may have been present at the moment of the accident, it is no longer present when the

Fig. 50.—Lateral view, plantar flexion.

patient comes under the surgeon's care or is sent for roentgenologic study; and at the latter time can only be presumed to have existed by certain signs, especially evidences of a sprain fracture or detachment of the anterior tubercle of the tibia (Fig. 30).

If, in addition to division of the anterior tibiofibular ligament, one divides also the feltlike interosseous ligament, then a separation of the fibula from the tibia almost to the distance of 3 cm. may be possible; but even with this amount of diastasis of the anterior border of the fibula from the tibia, these bones are still united by the posterior tibio-

fibular ligament, so that the fibula is not entirely freed from the tibia though its lower end has become so movable as easily to permit a fracture by flexion.

Section of the posterior inferior tibiofibular ligament, as, indeed, noted by Quénu [58] (1907) permits only an insignificant separation of the fibula and tibia; and Quénu thinks that in life rupture of this ligament never occurs—at any rate it seems certain that this ligament is never ruptured alone, but only in conjunction with rupture of the anterior and interosseous ligaments. It is to be noted furthermore that it is next to an unheard of thing for the astragalus to be separated from the external malleolus, owing to the almost indestructible posterior band of the external lateral ligament; so that diastasis is not produced by a wedgelike action of the astragalus described by so many writers. I know of no case of true ascent of the astragalus between the tibia and fibula unaccompanied by the external malleolus: even in the beautiful case of diastasis recorded by Millikin,[59] though it is truly said that "the astragalus was jammed up between the outer surface of the tibia and the unfractured fibula" yet the astragalus had not been detached from the fibula and, therefore, cannot have acted as a wedge in driving the bones apart, though it certainly acted as a prop to keep them asunder. I have been at pains to reproduce this lesion on the cadaver, demonstrating that it is quite possible for the astragalus to become lodged against the outer surface of the tibia, between the latter bone and the fibula, without rupture of the posterior band of the external lateral ligament; though, of course, it is necessary to divide the internal lateral ligament and the inferior (anterior and posterior) tibiofibular ligaments, as well as the interosseous ligament and the interosseous membrane (the latter as far up as the upper third of the leg). And unless, in addition to the above, the anterior and middle bands of the external lateral ligament were divided, the astragalus maintained itself only in very unstable equilibrium between tibia and fibula; though no doubt in life, the stability would be greater owing to muscular tension. Moreover, in the type of fracture with diastasis in which the fibular fragment is accompanied by a fragment detached from the external surface of the tibia (as in Cooper's illustration copied by Vidal de Cassis), and which is usually described as exhibiting ascent of the astragalus between the bones of the leg, or along the outer side of the tibia, the astragalus carries with it the external malleolus (the lower fibular fragment), so that in no true sense has the astragalus ascended between the bones. Of course, it is true that dislocation of the astragalus may occur, forward or backward, detaching it from the fibula; and there is no

58. Quénu: Rev. de chir. **35**:897, 1907.
59. Millikin: Ann. Surg. **69**:650, 1919.

denying the possibility of its being dislocated upward between the intact tibia and fibula. All that I contend is that for it to become detached from the external malleolus in fractures of the leg bones at the ankle must be extremely rare, as there does not appear to be any such case on record.[60]

The Posterior Marginal Fragment of the Tibia, and Posterior Displacement of the Foot.—The existence of this fragment as the sole osseous lesion in a certain number of cases now on record,[61] including one of our own, proves that it may be the earliest stage of a lesion involving the ankle joint. But as there never has been any displacement of the fragment in these isolated lesions, it cannot be considered of much importance unless associated with a fracture of the fibula. As already noted, the existence of this fragment as a complicating lesion was well known to Cooper; it was noted by Earle[62] (1828) and by

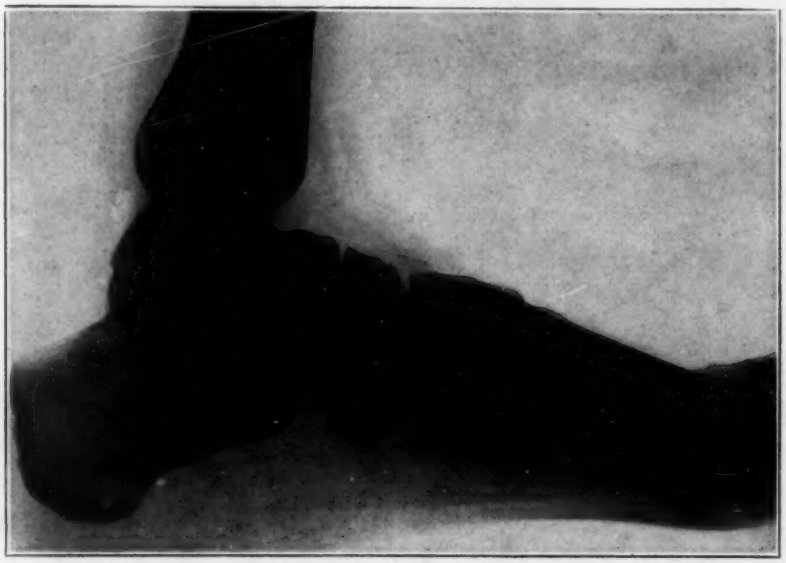

Fig. 51.—Lateral view, dorsiflexion.

60. Gross (System of Surgery, Ed. 5, Philadelphia **2**:85, 1872) says Druitt refers to such a case, but I have been unable to find the original report to ascertain the exact lesions. Wendel (Beitr. z. klin. Chir. **21**:146, 1898) collected five cases of upward dislocation without fracture; but the exact lesions do not appear to have been determined definitely in any case.

61. Tanton (Footnote 29, p. 165) refers to twenty cases recorded prior to 1916. As an isolated lesion it was first observed by Meissner (Beitr. z. klin. chir. **61**:136, 1909).

62. Earle: Lancet **2**:346, 1828-1829.

Adams [63] (1835); it was observed at necropsy by Dupuytren "with surprise" according to Malgaigne [64] (1832); Thaon [65] presented a necropsy specimen and said he had often seen this fragment in experiments made by Tillaux; it was clearly and accurately if succinctly described by Nélaton [66], (1847), and the fragment was recognized as a serious complication by Edmund Andrews [67] (1883, 1897). Since the introduction of the roentgen ray, it has been studied by Chaput [68] (1899, 1907), Bondet [27] (1899), Grashey [69] (1907), Meissner [61] (1907), Plagemann [70] (1911), Destot [8] (1911), Quénu (1912-1915), and by Stimson (in every edition of his book since 1899). Hence it was with surprise that surgeons who were tolerably familiar with the literature of their profession, as well as with fractures of the ankle, saw Cotton [71] (1915) describe it as "a new type of ankle fracture" which "has never been adequately described in print and has apparently escaped the notice even of those who deal with fractures habitually;" and noticed his complacent comment that in certain circles it was called "Cotton's fracture;" as well as his statement that he believed there were no necropsy specimens.[72] Cotton, however, did well to call attention to its frequency; as did Speed in a paper which was not published until after Cotton's paper was read, though it appeared in print before the latter.

The mechanism by which this fracture is produced is almost certainly, as contended by Lucas-Championnière,[73] a crushing force from below upward;[74] it is possible that traction by the posterior inferior

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63. Adams, in Todd: Cyclopedia of Anatomy and Physiology, London **1**:161, 1835-1836.

64. Malgaigne: Gaz. méd. de Paris **3**:647, 1832.

65. Thaon: Bull. Soc. anat. de Paris **45**:212, 1870.

66. Nélaton: Eleménts de path. chir., Ed. 2, Paris, **3**:296, 1874.

67. Andrews in Ashhurst: International Encyclopedia of Surgery, New York **3**:707, 1883; also in Internat. Clinics, Philadelphia, 1897.

68. Chaput: Bull. et mém. Soc. de chir. de Paris **25**:776, 1899; Les fractures malleolaires, Paris, 1907.

69. Grashey: Fortschr. a. d. Geb. d. Röntgenstrahlen. **11**:152, 1907.

70. Plagemann: Beitr. z. klin. Chir. **73**:688, 1911.

71. Cotton, F. J.: A New Type of Ankle Fracture, J. A. M. A. **64**:318 (Jan. 23) 1915.

72. In addition to the necropsy specimen which figures in Cooper's Plate XVII (reproduced here as Fig. 3), those described by Stimson and that of Thaon, already alluded to, there are in the Mütter Museum of the College of Physicians of Philadelphia three necropsy specimens showing this posterior marginal fragment. The most recent of these specimens has been in the museum for a period at least of forty years. (A description of these specimens will be published elsewhere by Dr. Bromer and myself.)

73. Lucas-Championnière: Bull. Soc. anat. de Paris **45**:212, 1870.

74. Rochet (Rev. d'orthop. **1**:269, 1890) produced it experimentally by dropping a weight of 60 kilograms, from a height, on the upper end of the tibia while the foot was in plantar flexion.

tibiofibular ligament, through the medium of the fractured lower end of the fibula, may aid in displacing the fragment, even if it cannot be the sole cause of its detachment. The size of the fragment varies from a small portion of the lip to a large fragment, extending 10 cm. up the posterior surface of the shaft. McKnight, at the meeting of the Philadelphia Academy of Surgery, May 2, 1921, showed a roentgenogram (anteroposterior view only) of a fracture which I believe conformed to this type, though the fragment was the largest with which I am acquainted: the fragment (an isolated lesion, without displacement) included nearly all the posterior lip of the tibia, as well as its entire lateral (fibular) border; and the apex of the large wedge extended to a point about 10 cm. above the articular surface, on the posterolateral border of the tibia.

TABLE 1.—INCIDENCE OF POSTERIOR MARGINAL FRACTURES

	Total Ankle Fractures	Posterior Marginal Fractures			
		Associated	Isolated	Total	Per Cent.
Ashhurst and Bromer	300	57	1	58	19
Chaput (Les fractures malléolaires, Paris, 1907)	136	42	0	42	30
Destot (Quénu: Bull. et mém. Soc. de chir. de Paris, **39**: 165, 1913)	1700	139	6	145	8.5
Quénu (Rev. de chir. **45**:260, 1912)	129	11	1	12	9.3
Sear (M. J. Australia, **1**:526, 1917)	156	26	3	29	18.6
Speed (Surg., Gynec. & Obst. **19**:73, 1914)	161	16	0	16	10

INCIDENCE OF ASSOCIATED POSTERIOR MARGINAL FRACTURES

Type	Ashhurst and Bromer		Destot	
	No.	Per Cent.	No.	Per Cent.
Fibula, oblique mixed	0	....	0	....
Low Dupuytren	51	88	89	64
Bimalleolar	0	....	17	12
Pott's	5	8.6	29	20
Adduction	1	1.7	0	....
Maisonneuve	0	....	4	3
	57		139	

The lesion corresponds, as Tanton [29] (loc. cit., p. 171) has noted, to Rhea Barton's fracture of the posterior margin of the radius.

It is a much more frequent complication than commonly supposed. Among our 300 cases it was present no less than fifty-eight times, or in 19 per cent. of all the cases; and in 51, or 50 per cent., of those conforming to the "low Dupuytren" type.

There is sometimes confusion between this posterior marginal fragment and the intermediate fragment ("third fragment of Tillaux"),

as remarked before.[28] The posterior tubercle of the tibia, which limits posteriorly the gutter for the reception of the fibula (Fig. 4), and to which is attached the posterior tibiofibular ligament, may be fractured by the pull of this ligament. It appears then in lateral roentgenograms as an infraction or sprain fracture, but does not involve the ankle joint. In the true marginal fractures, on the other hand, the line of fracture always extends into the tibial plafond; the fragment (contrary to what is said by Tanton) usually remains attached to the external malleolus

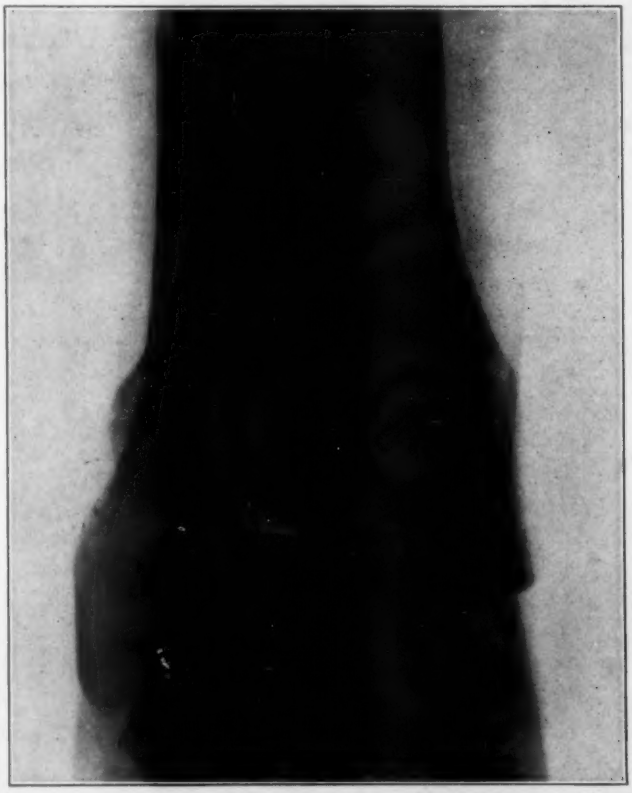

Fig. 52.—Rotation outward, obliterating outline of the posterior tubercle. (Same happens in mesial deviation of the tube.) The anterior tubercle overlaps the lateral margin of the fibula.

by the posterior tibiofibular ligament, and is often displaced backward with the external malleolus; sometimes (more frequently than thought) it may be detected in anteroposterior roentgenograms as a deltoid fragment; and, if large, its shadow sometimes overlaps that of the median border of the tibia, giving a double contour.

The posterior marginal fragment, as already remarked, may occur as an isolated lesion; only once in our series (Fig. 44). It may be

associated with (*a*) merely a diastasis, with sprain fracture of the anterior tubercle of the tibia (Fig. 31); (*b*) it may accompany the mixed oblique fracture of the fibula in the various stages of the fracture by outward rotation, being small (Fig. 32), medium sized (Fig. 33), or large (Fig. 34); and (*c*) it occurs also with fractures by abduction (Figs. 35 and 36), though very much less frequently than with external rotation fractures. Quénu has had one case in which fracture of the internal malleolus was the only other lesion, and another fracture of precisely the same type has come under our notice since our series of 300 cases was completed.

Its presence is not necessary to permit the occurrence of posterior displacement of the foot (Figs. 16 and 25), but certainly favors it. This posterior displacement is very rare without the posterior marginal fragment. Either a posterior marginal fragment must exist, or there must be rupture of the posterior inferior tibiofibular ligament. It is only a continuance of the force (now the weight of the body) after the fracture has been produced, that causes the displacement, since there are in our series no less than fourteen instances (out of a total of fifty-eight posterior marginal fractures) with slight if any displacement.

The factors which permit posterior displacement of the foot deserve a few words. Quénu was the first, I believe, to point out that the essential lesion is freeing the lower end of the fibula from the tibia; and I may remark that this necessity is merely a corollary of what has been insisted on so often in these pages, namely the indissolubility of the union between the astragalus and the fibula. I cut everything else at the ankle (tendons and ligaments as well as all other soft parts) leaving only the middle and posterior bands of the external lateral ligament attaching the external malleolus to the foot. Under these circumstances even an incomplete posterior dislocation of the foot cannot occur (except by rotation of the foot inward around the long axis of the leg, a mechanism which does not occur in life). I then cut the middle band of the external lateral ligament; but still no posterior displacement could be produced. Next I fractured the fibula 7.5 cm. (3 inches) above its tip; but this did not permit posterior displacement. Finally, I divided the anterior inferior tibiofibular ligament, and even the interosseous ligament; but so long as the fibula remained attached to the tibia, and the atragalus to the fibula, no posterior dislocation could be produced. On another foot, I divided all structures uniting the foot to the fibula except the middle band of the external lateral ligament; but no dislocation of the foot was possible; then I divided also the posterior inferior tibiofibular ligament and the interosseous ligament (which allowed less diastasis than when the anterior tibiofibular ligament was divided), but still no posterior dislocation of the foot was possible except by rotating it inward around the one remaining ligament as a pivot.

As a result of these investigations, it may be concluded that either the middle or the posterior band of the external lateral ligament is sufficient to hold the astragalus against the external malleolus, and that (even after fracture of the internal malleolus or rupture of the internal lateral ligament) *no backward dislocation of the foot can occur* (1) unless the astragalus is freed from the external malleolus by rupture of both the middle and posterior bands of the external lateral ligament (a lesion which apparently has not been recorded in association with

Fig. 53.—Rotation inward; very little change from the normal. (Same happens in lateral deviation of the tube.)

fractures at the ankle); (2) or unless the external malleolus is freed from the tibia (*a*) by diastasis of the inferior tibiofibular joint, with or without detachment from the tibia of an intermediate fragment; (*b*) by fracture of the fibula in such a way as to detach with the lower fragment of the fibula the fibular insertions of both the middle and posterior bands of the external lateral ligament (in other words, unless a virtual diastasis occurs, but one in which the intermediate

fragment belongs to the fibula instead of to the tibia). One very frequent fracture of the fibula (the mixed oblique of Destot) fulfils these requirements; as does another less usual, namely a transverse fracture of the external malleolus proper below the level of the tibia. But as the latter fracture usually is subperiosteal or incomplete, and without displacement, it follows that posterior displacement of the foot with this lesion is unknown.

Therefore, the fractures which may be accompanied by posterior displacements of the foot are those of the Pott or Dupuytren type, in which diastasis is the rule; and those of the low Dupuytren variety, in which the fracture of the fibula is of the mixed oblique type. Quénu [75] is wrong, I am sure, in claiming (1912) that the existence of a posterior marginal fragment is a necessary condition for the occurrence of a posterior subluxation (Figs. 16 and 25); but it is well to remember that the distorted shadows of a roentgenogram in a case of fracture of the low Dupuytren variety, in any of its stages, may mislead the observer into the belief that a posterior displacement is present, when the appearances are due entirely to an outward rotation of the astragalus around the long axis of the leg.

Forced Movements of Adduction (Tibial Flexion).—Since the time of Cooper and that of Maisonneuve and Bonnet, there has been little dispute about the mechanism of these fractures: it has been generally recognized that a tearing off of the external malleolus is the first lesion (Fig. 37), followed by a compression fracture of the internal malleolus (Figs. 38 and 39); or, when the weight of the body forces the tibia heavily on the displaced astragalus, a splitting upward of the tibial shaft occurs, the line of fracture commencing at some point of the articular surface, splitting this in the sagittal plane and terminating on the median border of the tibia at a variable distance above the internal malleolus (Figs. 40, 41 and 42). The more nearly longitudinal the line of fracture, the less necessary will it be for the external malleolus to be fractured as a preliminary step: such fractures verge into those due to comminution upward in the long axis of the limb. Stimson [76] (1912) gives an admirable illustration of such a longitudinal fracture of the tibia without any lesion of the fibula. But if the displacement in these longitudinal fractures is marked enough, the fibula may break secondarily (by flexion or torsion) above the inferior tibiofibular joint, as the lower end of the fibula is carried inward with the astragalus (Fig. 41). A variant of this type is the lesion recorded by Silhol [77] in which the fibular fracture is replaced by an intermediate

75. Quénu: Rev. de chir. **46**:1912.
76. Stimson: Fractures and Dislocations, Ed. 7, Philadelphia, 1912, Plate 27.
77. Silhol: Bull. et mém. Soc. de chir. de Paris **42**:819, 1916.

fragment detached from the tibia. Even in one of our own cases (Fig. 40), there is an intermediate fragment, in addition to the fracture of the fibula.

The supramalleolar fracture by adduction, produced experimentally by Tillaux, and already mentioned on page 71, may be considered the most advanced degree of this type (Figs. 42 and 43.)

SUMMARY

These then are the abnormal movements—external rotation, abduction, adduction—which are responsible for the great majority (95 per cent.) of fractures about the ankle: external rotation causes about 61 per cent., abduction about 21 per cent., and adduction about 13 per cent. of the lesions. The remaining small proportion (5 per cent.) consists chiefly of those fractures which may be recognized either as caused by compression in the long axis of the limb, or by direct crushes; or even by very rare forced movements such as straight flexion or extension, internal rotation, etc. The fractures by compression in the long axis of the limb include (*a*) the isolated fractures of the tibial margins (anterior, posterior [Fig. 44], median or even lateral), which Sear [78] speaks of as "vertical plane fractures;" (*b*) comminuted fractures of the tibial plafond (Fig. 45), and (*c*) T or Y-fractures involving the ankle joint (Figs. 46 and 47), which may be regarded as an advanced degree of the supramalleolar V-fractures described by Gosselin,[79] the latter usually being complicated by a fissure extending into the ankle joint, the mechanism being the same as in those of the radius with comminution of the wrist fragment.[80] The supramalleolar fractures of Malgaigne [81] (1847) are thus distributed according to their mechanism, some into those by adduction, others as due to compression in the long axis of the limb, and a few probably due to direct violence (Fig. 48). We count no supramalleolar fracture as one involving the ankle unless it falls within 4 cm. of the joint level (Richet, 1875). Fractures as close to the ankle joint as 4 cm. compromise its functions as surely as do supracondylar fractures of the humerus compromise those of the elbow.

Now, it is because of the impossibility of classifying together anatomically lesions which, as Stimson says, are merely alternative, or whose differences are due to the early cessation of the force before the typical form has been reached; or, I may add, to its continuance after the typical stage has been passed—it is because of this impossibility, I

78. Sear: M. J. Australia **1**:526, 1917.
79. Gosselin: Gaz. d. hôp. **28**:218, 1855.
80. Gosselin: Bull. et mém. Soc. de chir. de Paris **5**:147, 1863.
81. Malgaigne: Fractures, Paris, 1847, p. 818.

repeat, that I believe a classification based on mechanism, imperfect though it be, is, nevertheless, more easily understood and remembered. And it is for this purpose that Dr. Bromer and I have ventured to

TABLE 2.—Classification of Three Hundred Ankle Fractures

A. Fractures by External Rotation			
1. First Degree: Lower end of fibula only ("mixed oblique")		79	(26 %)
2. Second Degree: Same, *plus* rupture of internal lateral ligament or fracture of internal malleolus ("low Dupuytren")		100	(33 %)
Viz.,			
(*a*) Internal lateral ligament, uncomplicated	13		
Internal lateral ligament complicated by posterior marginal fragment of tibia	13		
(*b*) Internal malleolus, uncomplicated	32		
Internal malleolus complicated by posterior marginal fragment of tibia	42		
3. Third Degree: Same, *plus* fracture of whole lower end of tibia, representing the internal malleolus		5	(1.7 %)
Total Fractures by External Rotation		184	(61 %)
B. Fractures by Abduction (Fibular Flexion)			
1. First Degree: Internal malleolus only		20	(6.6 %)
2. Second Degree: Same *plus* fracture of fibula (transverse, above or below tibiofibular joint)		41	(13.7 %)
(*a*) Below inferior tibiofibular joint (no diastasis) ("bimalleolar fracture")	13		
(*b*) Above inferior tibiofibular joint (with diastasis) ("Pott's fracture," "Dupuytren type")	28		
3. Third Degree: Internal malleolus represented by whole lower end of tibia		2	(0.66%)
Total Fractures by Abduction		63	(21 %)
C. Fractures by Adduction (Tibial Flexion)			
1. First Degree: External malleolus only, transverse, at or below level of tibial plafond		27	(9 %)
2. Second Degree: Same, *plus*			
(*a*) Internal malleolus below level of tibial plafond ("bimalleolar fracture")	3		
(*b*) Median surface of tibia up and in from joint surface	8	11	(3.6 %)
3. Third Degree: Same, *plus* whole lower end of tibia ("supramalleolar fracture by adduction")		2	(0.66%)
Total Fractures by Adduction		40	(13.3 %)
D. Fractures by Compression in Long Axis of Leg			
1. Isolated Marginal Fractures	1		
2. Comminution of tibial plafond	3		
3. T or Y-fractures ("V-fractures of Gosselin")	4		
Total Fractures by Compression in Long Axis of Leg		8	(2.7 %)
E. Fractures by Direct Violence (Supramalleolar types)		5	(1.7 %)

arrange our series of fractures as shown in Table 2. Our aim has been to place under each mechanism, first, the simplest resulting form of fracture, and to advance thence to more complicated lesions,

noting at the same time in their appropriate places the variants and the complications of the simple or the more complex lesions which were encountered. It is true that it is not always easy to determine the mechanism, even with all the aid derived from the clinical history, the roentgen ray and a knowledge of the lesions which can be produced on the cadaver; but the more one studies the subject, the fewer exceptions he will find to the general laws of mechanics; and the more his

TABLE 3.—ANATOMOPATHOLOGIC CLASSIFICATION OF THREE HUNDRED ANKLE FRACTURES

A. Fibula, Below Inferior Tibiofibular Joint				
1. Alone (slight or no displacement, often subperiosteal)	27	(9 %)		
2. Same, *plus* internal malleolus below tibial plafond (includes bimalleolar fractures by adduction and by abduction)	16	(5.0 %)		
3. Same, plus split of tibia up and in	8	(2.6 %)	51	(17 %)
B. Fibula, Obliquely Through Inferior Tibiofibular Joint				
1. Alone (slight or no displacement, often subperiosteal)	79	(26 %)		
2. Same, plus rupture of internal lateral ligament	26	(9 %)		
3. Same, *plus* fracture of internal malleolus	74	(25 %)	179	(60 %)
C. Fibula, Above Inferior Tibiofibular Joint				
1. Alone (slight or no displacement, often subperiosteal)	0	(....)		
2. Same, *plus* rupture of internal lateral ligament and diastasis	8	(2.7 %)		
3. Same, *plus* fracture of internal malleolus and diastasis	20	(6.6 %)	28	(9.3 %)
D. Tibia, Involving Ankle Joint				
1. Internal malleolus alone (slight or no displacement, often subperiosteal)	20	(6.6 %)		
2. Isolated marginal fractures	1	(0.33%)		
3. Comminuted, T and Y-fractures	7	(2.3 %)	28	(9.3 %)
E. Supramalleolar Fractures, Not Involving Ankle Joint Directly	14	(4.3 %)	14	(4.3 %)

experience increases, the easier will it become to recognize the variants from the typical lesions. Those fractures which have given us most concern are the true bimalleolar fractures with little or no displacement, since these may be caused possibly by external rotation, certainly both by abduction and adduction. But as the total number of these fractures observed is small (sixteen cases or only 5 per cent. of the entire series), and as even among this number the mechanism was reasonably certain in all but ten cases (3 per cent. of the entire series), the margin of error is small.

TABLE 4.—Catalog of Three Hundred Fractures Involving the Ankle

	No. Cases	
A. By Rotation of the Astragalus Outward Around the Long Axis of the Leg		
I. First Degree: Lower end of fibula only. Oblique or spiral fracture from above and behind, down and front, the line of fracture passing through the inferior tibiofibular joint. Line varies from nearly transverse to nearly longitudinal, but is always higher on posterior than on anterior surface of fibula. In 84 per cent. of cases the lower anterior end of line of fracture was on anterior surface of external malleolus between its tip and the anterior inferior tubercle of the tibia; in 8 per cent. of cases it was above the anterior tubercle (when there was diastasis or the tubercle was detached); and in 8 per cent. it passed through the fibula at the level of the anterior tubercle on the tibia. Slight or no displacement	76	
Variant: fracture of fibula in *upper third* from lesion at ankle (Maisonneuve, 1840)	1	
(In this case there was incomplete fracture of fibula below its head (Fig. 17) but without bony lesion at ankle)		
Complication: intermediate fragment	2	
	—	79
II. Second Degree: Fibula as in first degree, *plus* rupture of internal lateral ligament or fracture of internal malleolus. There may be slight or considerable lateral displacement; but in uncomplicated cases there is rarely a real posterior displacement, the apparent posterior displacement seen in lateral roentgenographic views being due usually to outward rotation which distorts the shadows.		
(*a*) Internal Lateral Ligament (in roentgenograms this lesion is differentiated from first degree only by presence of lateral displacement of astragalus from internal malleolus with slight displacement of lower fibular fragment)	12	
Variant: Diastasis without fracture of fibula (Fig. 16)	1	
First Complication: Fracture of posterior tibial margin. Line of fracture usually extends about 2 cm. up along posterior surface of tibia, and running down thence nearly vertically, detaches a mere chip or more often one fourth to one third of the articular surface; rarely as much as one half	10	
Variant: Diastasis without fracture of fibula, but with posterior marginal fragment (Fig. 31)	1	
Second Complication: Intermediate fragment	0	
Third Complication: Second fracture of fibula in upper third (Maisonneuve's fracture; these two cases occurred in opposite legs of same patient)	2	
(*b*) Internal Malleolus	32	
(with slight or no displacement, twenty cases)		
First Complication: Fracture of posterior tibial margin (Figs. 32, 33, 34)	40	
(slight or no displacement, fourteen cases)		
(lateral and posterior displacement twenty-six cases)		
Second Complication: Intermediate fragment	2	
	—	100
III. Third Degree: Internal malleolus represented by whole lower end of tibia		5
(This will include most cases of separation of lower epiphysis of tibia)		
Total Fractures by Mechanism of Outward Rotation		184

TABLE 4.—Catalog of Three Hundred Fractures Involving the Ankle—(*Continued*)

	No. Cases
B. By Abduction (Fibular Flexion) of Foot	
I. First Degree: Fracture of internal malleolus only, transverse, at or below level of tibial plafond. No displacement	19
II. Second Degree: Rupture of internal lateral ligament or fracture of internal malleolus, as in first degree, followed by fracture of fibula more or less transverse either below or above inferior tibiofibular joint	
(*a*) Fracture of fibula below inferior tibiofibular joint (i.e., of external malleolus proper) with fracture of internal malleolus or rupture of internal lateral ligament	13
(with no displacement, three cases; internal malleolus displaced more than external, four cases; both malleoli equally displaced, six cases)	
Variant: Crush of calcaneum (replacing fracture of internal malleolus) followed by compression fracture of external malleolus	1
(*b*) Fracture of fibula above inferior tibiofibular joint (in lower third), the line of fracture indicating a fracture by flexion (Biegungsbruch), with slight or no torsion; slightly oblique, often comminuted with detachment of a wedge-shaped fragment from the surface of flexion, and its apex toward the surface of extension	28
	— 42
1. Fracture of fibula alone, or with rupture of internal lateral ligament	6
Variant: Diastasis without fracture of fibula	2
2. Fracture of fibula with fracture of internal malleolus at or below level of tibial plafond	12
First Complication: Posterior marginal fragment of tibia (Figs. 35 and 36)	5
Second Complication: Intermediate fragment	3
III. Third Degree: Internal malleolus represented by whole lower end of tibia (This will include some cases of separation of lower epiphysis of tibia)	2
Total Fractures by Mechanism of Abduction	63
C. By Adduction (Tibial Flexion) of Foot	
I. First Degree: Fracture of external malleolus, transverse, at or below level of tibial plafond. Slight or no displacement, often subperiosteal	26
Complication: Posterior marginal fragment of tibia (infraction)	1
	— 27
II. Second Degree: Fracture of external malleolus as in first degree, *plus:*	
(*a*) Fracture of internal malleolus below level of tibial plafond	2
Variant: Fracture of external malleolus represented by rupture of external lateral ligament	1
(*b*) Fracture of median surface of tibia up from tibial plafond	8
	— 11
III. Third Degree: Fracture of fibula usually above inferior tibiofibular joint, *plus* fracture across tibia above ankle joint (Tillaux, 1872)	2
Total Fractures by Mechanism of Adduction	40
D. By Compression in Long Axis of Leg	
I. Isolated marginal fractures of tibia: anterior margin, none; posterior margin, 1; median margin, none; lateral margin, none	1
II. Comminution of tibial plafond	3
III. T or Y-fractures of Tibia into Ankle-Joint	4
	— 8
E. Fractures by Direct Violence (Supramalleolar Types)	5
Grand Total	300

It will be noted that in our classification the fibular lesion dominates the clinical picture in the first two classes (those of outward rotation and abduction), and that these correspond to the first grand division of Destot's classification—those fractures in which the equilibrium of the foot is involved; while the third, fourth and fifth classes correspond to Destot's second division—those fractures which involve the tibial pestle and compromise the function of support. For the sake of completeness we also give an anatomopathologic classification, constructed on the same principles (Table 3).

Following the classifications is a catalog of the lesions encountered in this series of cases, which, with the aid of the classifications as a guide, we hope may prove of interest to students of fractures (Table 4).

ROENTGEN-RAY STUDY OF THE ANKLE JOINT

By Dr. Bromer

I. THE NORMAL ANKLE JOINT—ANATOMY

By means of the roentgen ray, the structure of the ankle joint can be most satisfactorily demonstrated. The tibiofibular mortise is shown (Fig. 49), and below it the trochlea of the astragalus fitting into it as a tenon. The "plafond" and the "cheeks" of the mortise are easily recognized, likewise the longer external malleolus and the anterior and posterior tubercles of the fibular groove. In the lateral view (Fig. 50), the low projection of the posterior lip, called by Destot the posterior malleolus, reinforcing the mortise posteriorly, can easily be visualized; and the point where it meets the trochlear surface of the astragalus, acting as a check on the latter when the foot is in plantar flexion as in walking, is most noticeable. (The degree of plantar flexion in the roentgenogram is much greater than is assumed in walking, but is used more fully to demonstrate the check). In this view, the longer external malleolus is shown overlapping the shadow of the internal malleolus. With stereoscopic plates they usually can be fairly well distinguished. The lateral tubercle on the posterior surface of the astragalus (os trigonum) is the farthest posterior shadow of the astragalus. In the interpretation of roentgenograms of the ankle joint, due care must be exercised not to diagnose a sprain-fracture of the posterior tubercle, where an accessory bone, the os trigonum, often is found. In general, no fracture exists, and it is this bone that is present if the surfaces of the fragment are smooth and rounded.

II. FUNCTION AND MOVEMENTS OF THE ANKLE JOINT

The roentgen ray can be used to demonstrate the movements of the ankle joint. Thus the rotation of the astragalus about the axis previously described by Dr. Ashhurst in full dorsiflexion and plantar flexion

is shown in Figures 50 and 51. The movements of the fibula were shown in the following way: A normal ankle was examined; the ankle and leg of the subject being securely bound to the table, a Bowen stereoscopic plate holder was placed beneath the ankle joint. Lateral views were made first with the foot in full dorsiflexion, then later with the foot in full plantar flexion. Great care was exercised that the position of the leg—i.e., tibia and fibula—was not altered. The stereoscopic plate holder gave an exact duplicate position for the second exposure. The tube was in no way shifted. Anteroposterior views were taken in the same way. In this plane, the heel was allowed to project as far as possible over the edge of the plate holder in order to obviate any change in height of the tibia above the plate due to change of position of the os calcis. The superior tibiofibular articulation was examined in the same way.

It was found that these views could be superimposed over diffused strong light. As an additional check the distances between the same relative points were accurately measured. The fact that the images of the tibia could be accurately superimposed would tend to rule out any possibility of error due to change in height of tibia. It was found that in full plantar flexion in the anteroposterior view the fibula moved inward and downward 1 mm. or, vice versa, so much expansion and movement upward occurred in full dorsiflexion. No change of the fibula in relation to the tibia could be found in the lateral views.

With regard to the upper extremity of the fibula, the forward movement of the fibula in plantar flexion was shown in the lateral view by an increase of 1 mm. in the distance between the posterior lip of the upper extremity of the fibula and the posterior border of the tibia. In the same way in the anteroposterior view the head of the fibula was found lying farther in toward the midline with the foot in plantar flexion than it was with the foot in dorsiflexion. All of these measurements were made by means of calipers, and the same level was obtained by means of superposition of the films over diffused strong light.

III. SOME DIFFICULTIES ENCOUNTERED IN INTERPRETATION OF THE ROENTGENOGRAMS OF THREE HUNDRED CASES

During the intensive study of the roentgenograms, from the point of view of roentgen interpretation, most interesting questions arose, necessitating definite solutions before any classifications or any theories of mechanism could be determined. These questions came almost entirely from a lack of standardized technic. The 300 cases had been examined by four different roentgenologists. The technic of only one of them was known. Immediately, the question arose: Does not this, or that, represent some variation from the real normal, produced by variations

in the procedures of these different roentgenologists, rather than a pathologic condition?

Textbooks on roentgenology describe certain more or less standardized positions for examination of the ankle joint. They point out anatomic landmarks above which to center the target of the tube. But none of them describe the variations in shadow which may occur: for instance, from increased or decreased target plate distance, or from variations produced by lateral shifting of the target, or by variations in posture of the joint. It would seem that opportunities for error are numerous. The very nature of hospital dressings—so different in the various institutions—constitutes one of the most prolific sources of error. The difficulty of exact centering through a plaster-of-Paris dressing or of an ankle encased in a fracture box is obvious.

The discussion which follows may seem to be far-fetched to some and futile to others. I am well aware of the conditions under which many busy roentgenologists work. I realize that a plate or film placed under an ankle with the target at any given distance above it, and the perpendicular assumed or guessed at, with a proper exposure technic will result in a so-called excellent negative. We admit that in a busy hospital service most of our patients were so examined. We admit that to the best of our knowledge no patients in any way afterward suffered, clinically, from such unstandardized examination at our hands. It is entirely probable that many clinicians can and will say, Why more? But I contend that in our study of these cases we were greatly hampered by such methods in arriving at definite conclusions. Gross lesions are usually apparent and easily diagnosed. The fine points in diagnosis are the difficult ones. It is only by exact methods that the science of diagnosis is advanced and the sum total of our knowledge of the subject increased.

The first difficulty that arose, the solution of which was of utmost importance, was the exact determination of just what on the roentgenogram determines diastasis of the lower tibiofibular junction. The normal ankle joint was roentgenographed under all possible conditions, in an effort to study the possible variations in shadows of the normal due to changes in technic. It was first examined at various target plate distances with the foot and target in the perpendicular plane; then the same ankle was roentgenographed with the leg in different angles of rotation and also with the foot in full dorsiflexion and plantar flexion, and again with the target deviated to either side from the perpendicular plane. A study of these results shows the necessity for the roentgenologist's deciding on one target plate distance for all examinations, also for him to formulate or design some scheme for quickly securing in each case the same perpendicular plane of the ankle and target,

especially in the anteroposterior view. A long upright rod with another at right angles, attached to its lower end, can readily be made and used for this purpose (goniometer).

From the results obtained on the roentgenograms showing the variations in target plate distance, it can be assumed that shadows on roentgenograms follow certain rules. Thus, as this distance decreases, all shadows proportionately increase provided there is no shift of the target in any direction, or any change in the position of the ankle, as, for instance, rotation of the limb. Thus the distance between the shadow of the line of the posterior tubercle on the fibular groove and that of the mesial margin of the fibula is increased if the target is moved nearer the plate, but likewise the width of the fibula and tibia seems to be proportionately increased. However, by no possible manipulation of the tube to either side up to 75 degrees from the perpendicular, or by inward or outward rotation of the leg to 80 degrees can the shadow of the most lateral point of the anterior tubercle be made to pass to the median side of the shadow of the median border of the fibula. (It seemed that the above-mentioned angles were the limits of the possible errors that could have been made in the roentgenograms of our series). Hence it was felt that we could safely say that whenever this did happen, tibiofibular diastasis was established. In fact, if the space between the lateral margin of the fibula and the lateral border of the anterior tubercle exceeds more than two thirds of the width of the fibula, it is most probable that the first degree of diastasis (Fig. 36) exists. This would seem to be certainly the case if the roentgenogram was made with the foot and ankle and tube in the absolute perpendicular plane (Figs. 49, 52 and 53).

Chaput [39] (1912) gave figures in millimeters showing variations of the "clear space" as he called it between the line of the posterior tubercle and the mesial border of the fibula, and claimed that if this space exceeded more than 3 mm., or if the area of the overlapping shadows of fibula and anterior tibial tubercle was less than 10 mm., diastasis was present. The objection to this as compared with the above method is obvious. Measurements on the roentgenogram vary greatly with changes in technic, target plate distance, etc., with age periods and in the different sexes. So a method establishing a means of estimation proportionate for each individual case is manifestly the best.

The larger the clear space between these bones, the greater the degree of diastasis. If there is additional roentgen evidence of a sprain fracture or detachment of the anterior tubercle, there is double assurance (Figs. 30 and 31). But here again one must make due allowance for the fact that when the patient reaches the roentgenologist reduction often has been established. The clear space will then not be abnormally

wide, and diastasis can only be presumed to have existed by the evidence of a sprain fracture or a fracture of the anterior tubercle. If these are not present, then no diagnosis of diastasis can be made by the roentgen ray. It seems possible that by a very complete study of normal ankles in both sexes, and in all age periods under identical conditions of technic, more absolute criteria can be established whereby this diagnosis can be made.

The clear space between the internal malleolus and the astragalus in the anteroposterior view is also a most interesting study. What determines the earliest or first degree of lateral displacement of the astragalus? This certainly is of the utmost importance in the determination of such displacement, particularly the so-called outward displacement. It was found that a 10 degree inward rotation of the leg increased this clear space, that the same amount of external rotation correspondingly decreased it; that variations in target plate distance proportionately increased or decreased it as the case might be, that tibial and fibular flexion of the foot both decreased it. So if a minute, careful diagnosis is to be made, the absolute perpendicular must be maintained. Had we had all roentgenograms of the 300 cases studied, made under identical technic, such diagnoses could have been made. We feel that quite a considerable degree of displacement had to be present, in fact so much that it would be apparent under any condition, any position of tube or ankle, before we could say definitely in the foregoing cases that a lateral dislocation existed. Probably also a better idea of rupture of the internal lateral ligament can be obtained by means of this clear space. Thus, when the internal malleolus is intact in the pure abduction fractures, this space may afford an idea of whether or not the internal lateral ligament has ruptured. Here again faulty methods prevented exact conclusions, and this again emphasizes the possibility of study under standardized conditions.

Study of the fibular groove led to the definite conclusion that the anterior tubercle always casts the more lateral shadow, the posterior tubercle the more mesial. This was confirmed by placing pieces of lead of different sizes on the lips of the groove in a living subject and then roentgenographing the ankle. It was noted, in the negatives made with deviations of the tube and change in position of the ankle, above described, that in the perpendicular plane the line of the posterior tubercle was always clearly defined, that this clear definition persisted when the leg was rotated in or with lateral deviation of the target; but with the leg rotated out or with mesial deviation of the target, this line had a tendency to disappear, becoming merged with the dense shadow of the thicker middle portions of the tibia. It is apparent that such variations may materially interfere with the exact determination of

just what constitutes the so-called posterior fragment. It may also interfere in the same way with recognition of the intermediate fragment. If this line remains intact, the posterior tubercle is not injured and the posterior inferior tibiofibular ligament is also probably not affected. It is possible that a deltoid shaped posterior marginal fragment broken from the tibia at a point mesial to and behind the posterior tubercle may be so displaced that its shadow overlaps the line of the posterior tubercle. The exact portion fractured can then only be determined by means of stereoscopic plates, and even then this is sometimes impossible. A fracture with such displacement was produced on a dried specimen, likewise another involving the posterior tubercle so as to interfere with the line of the latter on the roentgen-ray negative. In the anteroposterior view, they were indistinguishable, both appearing to be fractures of the posterior tubercle. However, stereoscopic films showed quite plainly the character of the first, i. e., a fragment displaced behind the tubercle and really not involving it.

In conclusion, this study has shown that while the roentgen diagnosis of the gross lesions of the ankle joint is a comparatively easy matter for the trained roentgenologist, the finer points of diagnosis so necessary to a thorough understanding of the mechanism are entirely dependent on more exact methods of technic than are often now employed.[82]

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82. In addition to the footnotes already given, the following, selected from more than 250 articles studied in the preparation of this paper, will be found of interest:

Bruns: Die Lehre von den Knochenbrüchen, Stuttgart, 1887, p. 57.

Farabeuf: Précis de manuel operatoire, Paris, 1909, p. 835.

Hamilton: Practical Treatise on Fractures and Dislocations, Philadelphia, 1860, pp. 443 and 685.

Messerer: Ueber Elasticität und Festigkeit der Menschlichen Knochen, Stuttgart, 1880, Plate 14, Figures 7, 11 and 3.

Quénu: Bull. et mém. Soc. de chir. de Paris **32**:943, 1906; Rev. de chir. **36**:62, 1907; Bull. et mém. Soc. de chir. de Paris **38**:1070, 1912; ibid. **45**:1142, 1919.

Scudder: Treatment of Fractures, Ed. 8, Philadelphia, 1915, p. 545.

Tillaux: Traité d'anatomie topographique, Paris, Ed. 2, 1878, p. 1023; Gaz. d. hôp. **59**:89, 1886.

STUDIES IN EXHAUSTION: III. EMOTION

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Intense emotion, especially fear, is one of the most injurious of human experiences. In fact, fear may be defined as the typical emotion, since practically every emotional state is allied to it, even anger, which must have originated in the impulse of self-protection against threatened danger. In the hope of discovering which organs participate in the production of fear, and the rôle assumed by each, I began a research to this end in 1908, the earlier results of which were reported in the Ether Day Address at the Massachusetts General Hospital, in 1910. In its various phases, this research has continued to the present time in the laboratory and the clinic, the emotive experiences of the war adding an overwhelming amount of human data to laboratory findings.

As stated in the preceding report,[1] it is practically impossible to formulate any experiment so that but one stimulus will act upon the animal; but as in the case of hunted foxes or of struggle in other animals, extreme exertion is the dominating activation, so in the case of a small animal subjected to threatened attacks, fear is the prevailing stimulus.

Most of our experiments were with rabbits which for varying periods of time were threatened by muzzled dogs. In some instances, the frightened rabbits were killed at once; in others, varying periods of time were allowed to elapse before death, that the later effects of the emotion might be studied; in still other cases, the animals were subjected to daily periods of fright on several successive days.

A. Histologic Studies

Rabbits.—Group 1: Experiments in which the rabbits were subjected to but one period of fright and then immediately killed.

Group 2: Experiments in which rabbits were subjected to but one period of fright, but were not killed until after two and one-half to six hours had elapsed.

Group 3: Experiments in which the rabbits were subjected to one period of fright on each of several successive days and killed shortly after the last period.

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1. Crile, G. W.: Studies in Exhaustion: II. Exertion, Arch. Surg. **3**:116 (July) 1921.

Differential Purkinje cell counts, made by Dr. Austin and Dr. Hitchings, from the rabbits in each of these groups, are shown in Tables 1, 2, 3 and 4. These findings are graphically represented in Figure 1. Equally marked changes were observed in the cerebellum and the cerebrum; less marked changes were found in the medulla, and changes even less marked than the latter were present in the spinal cord.[2]

Woodchucks.—While these experiments with rabbits were in progress, we had an opportunity to secure two woodchucks, which were dug out of their hibernating holes without being awakened. One was killed immediately; the other was frightened by several dogs for fifteen minutes without being injured in any way, and was killed six hours later.

The result of the comparative examination of the brains of these two animals is given in Table 5. The contrast is of special significance since it not only shows the possible effects of fear on the central nervous system, but the Purkinje cell counts of the woodchuck which was killed without being awakened from his hibernating sleep as compared with the normal Purkinje cell counts in dogs, in foxes and in rabbits (Table 6), which demonstrate strikingly the restorative and protective value of sleep. The sections of the brain of this hibernating woodchuck contained the most perfect cells of any among the thousands of sections examined in these researches on exhaustion.

In the experiments thus far described, histologic studies were made only of the nervous system. Later, after we had extended our histologic studies to include all the organs and tissues of the body, another series of experiments on fright were performed which demonstrated brain cell changes of the same nature and degree as those in the earlier series, and less marked, but frequent, changes in the cortex of the suprarenals and of the liver (Figs. 2 to 10). These findings are shown in Tables 7 and 8. The other organs and tissues showed no demonstrable histologic lesions (Table 9).

The effects of insomnia and sleep on the histologic changes produced by fright are shown in Table 10; and the findings in preliminary observations of the effects of sodium bicarbonate and of magnesium sulphate on the changes produced by fright are shown in Table 11.

B. Physicochemical and Functional Studies

Effect of Fear on Suprarenal Output

Thirty-five tests of the effect of fright on the suprarenal output were made in our laboratory by Drs. Hitchings, Sloan and Austin

2. Crile, G. W.: Proc. Soc. Exper. Biol. & Med. **7**:87-88, 1909-1910.

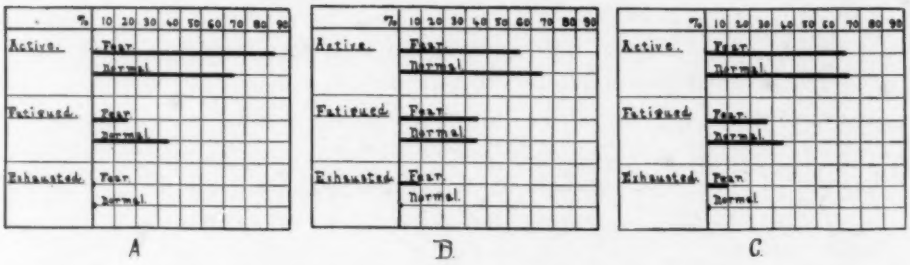

Fig. 1.—Average differential Purkinje cell counts in rabbits: *A*, rabbits killed immediately after a single period of fright; *B*, rabbits killed in from two and one-half to six hours after a single period of fright, and *C*, rabbits killed after daily periods of fright extending from two to three weeks.

TABLE 1.—Differential Counts of the Purkinje Cells of Rabbits Killed Immediately After One Seance of Fright

Duration of Fright	Stages		
	Active, per Cent.	Fatigued, per Cent.	Exhausted, per Cent.
60 minutes	73	25	2
60 minutes	84	16	0
25 minutes	69	28	3
25 minutes	94	5	1
10 minutes	77	21	2
60 minutes	86	14	0
30 minutes	87	12	1
25 minutes	83	17	0
35 minutes	92	8	0
40 minutes	89	11	0
Average	83.4	15.7	0.9
Normal average rabbit	64.8	34.5	0.7

Average duration of fright: 37 minutes

TABLE 2.—Differential Counts of the Purkinje Cells of Rabbits Killed at Varying Periods After One Seance of Fright

Duration of Fright	Interval of Time Until Killed, Hours	Stages		
		Active per Cent.	Fatigued, per Cent.	Exhausted, per Cent.
40 minutes	2¼	48	35	17
45 minutes	3½	70	26	4
90 minutes	3½	47	45	8
90 minutes	6	63	31	6
45 minutes	6	50	40	10
Average, 63 minutes	...	55.6	35.4	9.0
Normal rabbit average	...	64.8	34.5	0.7

TABLE 3.—Differential Counts of the Purkinje Cells of Rabbits Killed After Repeated Periods of Fright

Periods of Fright	Interval of Time Until Killed	Stages: Active, per Cent.	Fatigued, per Cent.	Exhausted, per Cent.
16 times in 3 weeks.....	Found dead*	59	27	14
14 times in 2 weeks.....	24 hours after last period	68	26	6
Average.....	.....	63.5	26.5	10.0
Normal rabbit averages...	.....	64.8	34.5	0.7

* This animal could not have been dead more than twelve hours, and the brain was therefore examined in a shorter time after the last period of fright than in the case of the second rabbit in this group, hence the greater percentage of exhausted cells could not be attributed to postmortem changes.

TABLE 4.—Comparison of Averages in Tables 1 to 3

	Active, per Cent.	Fatigued, per Cent.	Exhausted, per Cent.
Group I. Rabbits killed immediately after one seance of fright.....	83.4	15.7	0.9
Group II. Rabbits killed in from 2½ to 6 hours after one seance of fright.....	55.6	35.4	9.0
Group III. Rabbits killed after repeated seances of fright.....	63.5	26.5	10.0
Normal average.....	64.8	34.5	0.7

TABLE 5.—Comparative Study of the Brain of a Hibernating and of a Frightened Woodchuck

A. Cerebrum

Hibernating Woodchuck	Frightened Woodchuck
Cells moderately hyperchromatic with very even distribution of the granules	Marked chromatolysis, cells more irregular in outline than in hibernating woodchuck Many cells with large vacuoles. No hyperchromatic cells, some active, nearly all fatigued, or exhausted

B. Cerebellum

	Differential Purkinje Cell Count: Active, per Cent.	Fatigued, per Cent.	Exhausted, per Cent.
Hibernating woodchuck.....	90	10	0
Frightened woodchuck.....	4	58	38

TABLE 6.—Comparison of the Average Normal Purkinje Cell Counts in Dogs, in Foxes and in Rabbits with the Purkinje Cell Count in a Hibernating Woodchuck

	Differential Purkinje Cell Counts: Active, per Cent.	Fatigued, per Cent.	Exhausted, per Cent.
Normal dog.....	71.2	28.1	0.7
Normal fox.....	71.0	28.5	0.5
Normal rabbit.....	64.8	34.5	0.7
Hibernating woodchuck.....	90	10	0

TABLE 7.—Histologic Studies of Fear

Animal	Duration of Fright	Brain	Suprarenals	Liver
Rabbit	30 minutes	Cerebellum: occasional hyperchromatic and active cell; many fatigued; few exhausted Cerebrum: occasional hyperchromatic and active; almost all fatigued	About normal; only few cells in outer portion of cortex show loss of cytoplasm or vacuolation; medulla pale	Marked loss of cytoplasm and vacuolation in all cells
Rabbit	30 minutes	Cerebellum: no hyperchromatic cells; some active; almost all fatigued; few exhausted Cerebrum: occasional hyperchromatic cells; some active; almost all fatigued; few exhausted	Almost normal; very few cells of cortex show loss of cytoplasm; medulla deeply stained; no loss of cytoplasm in cells	Total loss of cytoplasm and complete vacuolation of all cells
Rabbit	30 min. per day for 45 out of 48 consecutive days	Cerebellum: active, 75%; fatigued, 22%; exhausted, 3% Cerebrum: some hyperchromatic, many exhausted	Cortex: only a few slightly affected cells Medulla: normal	Almost all cells show great loss of cytoplasma, only a few around central vein being in fair condition
Rabbit	20 min. per day for 45 out of 48 consecutive days	Cerebellum: active, 74%; fatigued, 24%; exhausted, 2% Cerebrum: more hyperchromatic than in the foregoing; some fatigued	Cortex: great loss of cytoplasm and vacuolation Medulla: lightly stained; otherwise normal	Every cell had lost almost all its stainable material

TABLE 8.—Histologic Studies of Fear

Animal	Duration of Fright	Brain	Suprarenals	Liver
Woodchuck	Frightened for 60 min. immediately after hibernation sleep; killed 5½ hours after termination of fear	Cerebellum: mostly fatigued; occasional exhausted; no hyperchromatic	Cortex: normal Medulla: normal	Almost entirely normal; only slight loss of cytoplasm and vacuolation around periphery of some lobules
Woodchuck	Had been in laboratory for some time; frightened for 15 min.; killed immediately	Cerebellum: many fatigued; occasional hyperchromatic Cerebrum: hyperchromatic and active	Cortex: only a narrow strip of outer cortical cells show slight loss of chromatin and vacuolation Medulla: normal	Slight uniform loss of cytoplasm

TABLE 9.—EFFECT OF FRIGHT ON VARIOUS ORGANS AND TISSUES OF THE BODY

	Belgian Hare, Male, Wt., 3,780 Gm.	Rabbit, White Female, Wt., 3,562 Gm.	Woodchuck, Male, 3 Years Old	Woodchuck, Male
Duration and manner of fright	Frightened twice a day for 20 minute periods for 47 days	Frightened twice a day for 20 minute periods for 47 days	Dug from hibernating hole; worried by dog for 1 hour; killed 4 hours later	Had been in laboratory some time; frightened for 15 minutes; killed immediately
Cerebrum	Some hyperchromatic cells, many fatigued	Some hyperchromatic cells, some fatigued; more hyperchromatic cells than in foregoing experiment	Most all cells active; occasional fatigued cell	Hyperchromatic and active
Cerebellum	75% active cells 22% fatigued 3% exhausted	74% active cells 24% fatigued 2% exhausted	No hyperchromatic cells; a few active; occasional exhausted cell	Occasional hyperchromatic cell, many fatigued
Liver	Nearly all cells show great loss of chromatin; only a few cells around central vein are in fair shape	Stainable material almost entirely lost; in worse condition than in preceding experiment	Almost entirely normal; slight loss of cytoplasm and vacuolation around periphery of some lobules	Almost normal; very slight loss of cytoplasm, uniformly distributed
Suprarenal	Cortex: a few slightly abnormal cells Medulla: deeply stained and normal	Cortex: many cells greatly affected, only a little stainable material left Medulla: less well-stained than in foregoing experiment	Cortex and medulla: entirely normal and well-stained; no cells show loss of cytoplasm	Nearly all cells normal; a narrow strip of outer cortical cells shows slight loss of chromatin and vacuolation
Kidney	.....	Bowman's capsule, tubules and glomeruli normal	.....	Bowman's capsule, tubules and glomeruli normal, well-stained
Thyroid	.....	Acini large; epithelium flat; no increase in interstitial tissue	Acini irregular; very large amount of interstitial tissue between acini	
Thymus	Faintly stained; medulla large; corpuscles numerous and small	Normal, well-stained; Hassell's corpuscles few		
Spleen	Normal	Normal	Normal and well-stained; few corpuscles and small; lymph spaces congested with red blood cells; fewer lymphocytes than normal	Normal, many well-stained lymphocytes
Lung	.....	Normal, epithelium intact	Greatly congested; alveoli not distended	Normal
Heart	Normal, faint striations	Normal, faint striations	Normal	Nuclei well stained; striations not present in section
Aorta	Normal	.....	Normal	
Voluntary muscle	.....	Normal, striations marked	Well stained and normal, beautiful striations	Normal
Lymph nodes	.....	.....	Normal; but lymph spaces extremely distended	Normal, crowded with lymphocytes

TABLE 9.—Effect of Fright on Various Organs and Tissues of the Body.—(Continued)

	Belgian Hare, Male, Wt., 3,780 Gm.	Rabbit, White Female, Wt., 3,562 Gm.	Woodchuck, Male, 3 Years Old	Woodchuck, Male
Stomach	.....	.....	Both acid and peptic cells well stained, muscle normal	Mucosa pink; acid, peptic, and parietal cells poorly defined
Intestines	Normal	Normal	Epithelium and muscle normal	Epithelium and muscle normal
Testicle	Much distorted and shrunken; well-stained cells in all stages of activity and fatigue	.....	Very few dividing cells or spermatozoa in tubules; very great number of large interstitial cells	Normal, active spermatogenesis; large number of interstitial cells crowded between tubules
Uterus	.....	Epithelium and cilia normal; muscle normal; not deeply stained		

TABLE 10.—Histologic Studies of Fear

Animal	Description of Experiment	Brain	Suprarenals	Liver
Rabbit	½ hour fright followed by insomnia 10 hours; killed immediately after insomnia period	Cerebellum: no hyperchromatic; few active; very marked fatigue in almost all cells Cerebrum: many fatigued; some hyperchromatic and active cells	Cortex: normal; no loss of cytoplasm	Stain not deep; cells all normal
Rabbit	½ hour fright followed by insomnia 10 hours; killed immediately after insomnia period	Cerebellum: few active cells; no hyperchromatic. almost all badly fatigued Cerebrum: some hyperchromatic and active cells; many fatigued cells	Slight loss of cytoplasm and vacuolation in some cells of cortex Medulla: deeply stained	Stain faint with uniform but not marked loss of cytoplasm; no vacuolation
Rabbit	30 minutes fright; insomnia 5 hours; sleep 5 hours; killed immediately	Cerebellum: mostly fatigued; no hyperchromatic Cerebrum: all fatigued; no hyperchromatic	Cortex: normal Medulla: normal	Normal
Rabbit	30 minutes fright; insomnia 5 hours; sleep 5 hours; killed immediately	Cerebellum: mostly fatigued; some hyperchromatic in stage II Cerebrum: mostly hyperchromatic	Cortex: normal Medulla: normal	Uniform loss of cytoplasm; some vacuolated cells; a few cells with eccentric nuclei; cell outlines regular

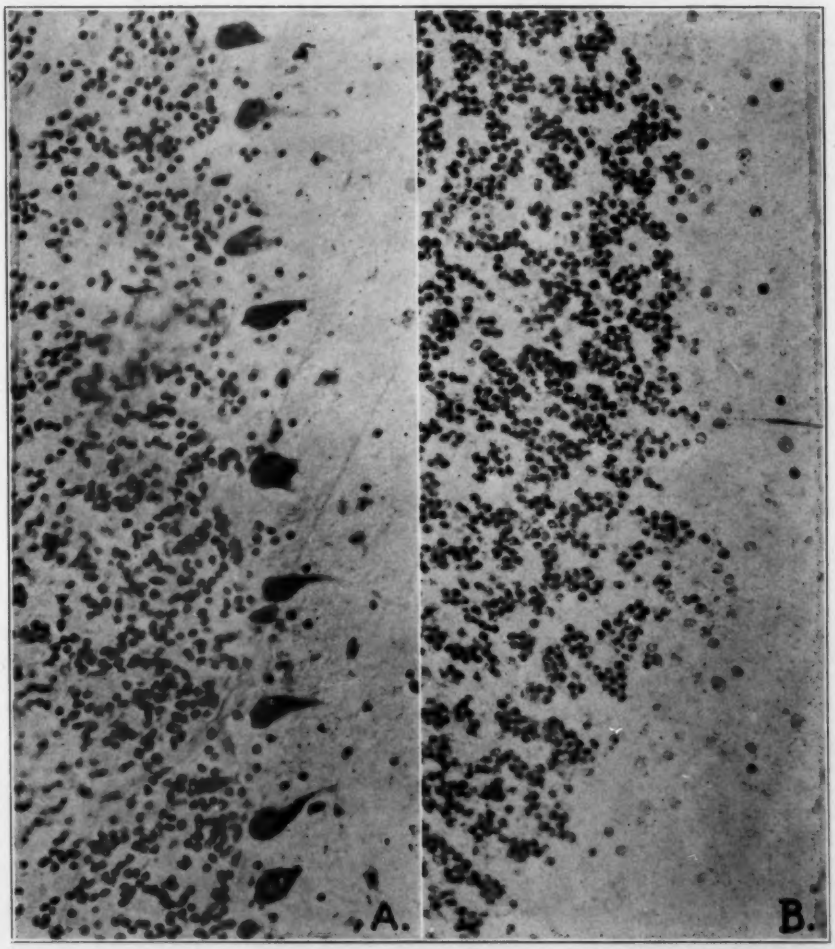

Fig. 2.—Effects of fright on the cerebellum of a rabbit (reduced from photomicrograph, × 310): *A*, section of normal cerebellum of a rabbit; *B*, section of the cerebellum of a rabbit after repeated periods of fright.

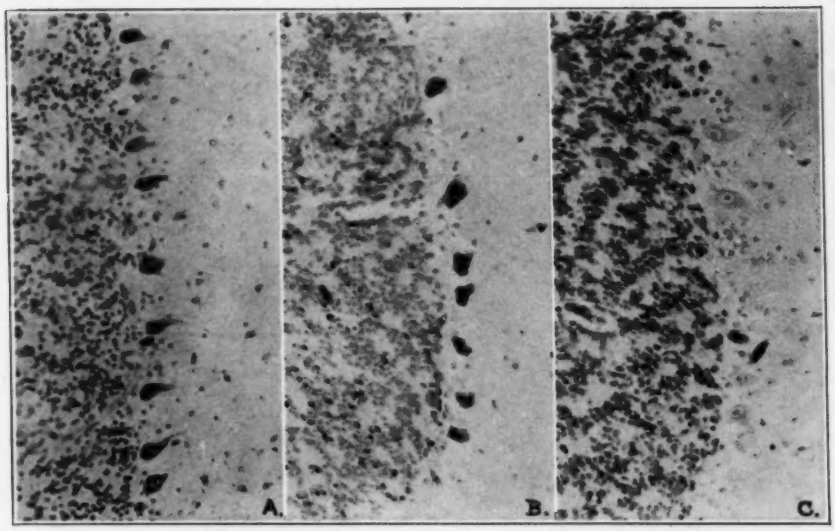

Fig. 3.—Immediate and late effect of fright on the cerebellum of woodchuck (reduced from photomicrographs, × 310): *A*, section of cerebellum of a hibernating woodchuck; *B*, section of cerebellum of a woodchuck killed immediately after a single period of fright; *C*, section of cerebellum of a woodchuck killed four hours after a single period of fright.

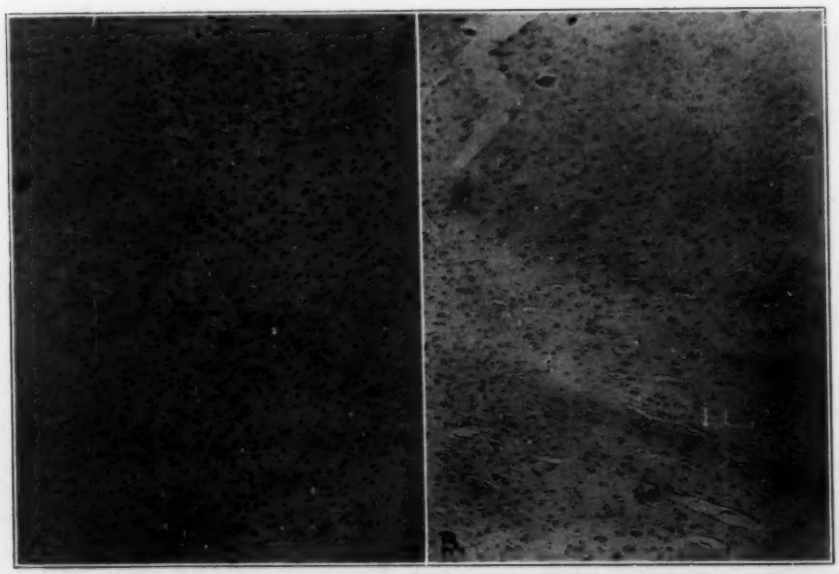

Fig. 4.—Effect of fright on the cerebrum of a rabbit (reduced from photomicrographs, × 85): *A*, section of normal cerebrum of a rabbit; *B*, section of cerebrum of a rabbit after repeated periods of fright.

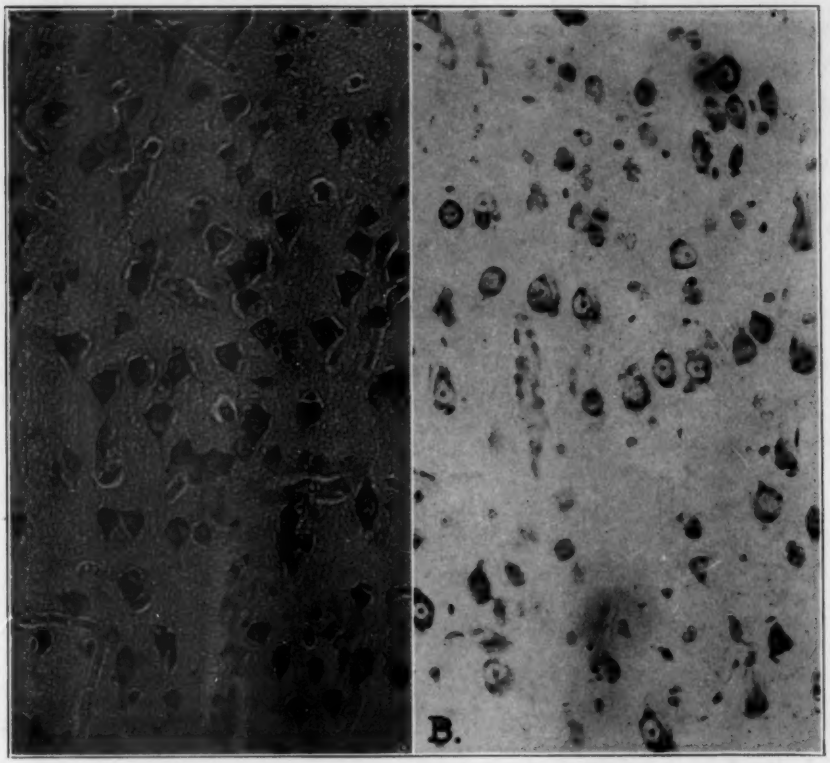

Fig. 5.—Effect of fright on the cerebrum of a woodchuck (reduced from photomicrographs, × 310): *A*, section of cerebrum of a hibernating woodchuck; *B*, section of cerebrum of a woodchuck killed four hours after a single period of fright.

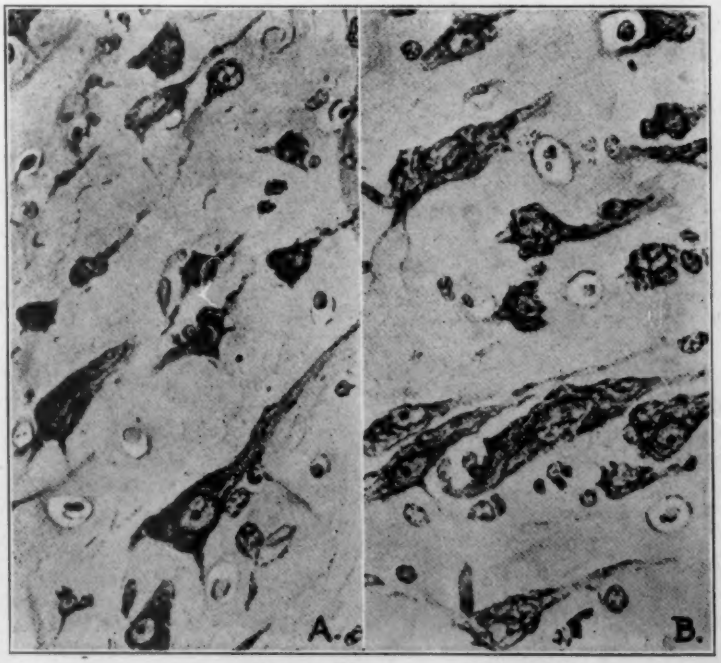

Fig. 6.—Effect of fright on the medulla of a woodchuck (from camera lucida drawings): *A*, section of medulla of a hibernating woodchuck; *B*, section of medulla of a woodchuck after a single period of fright.

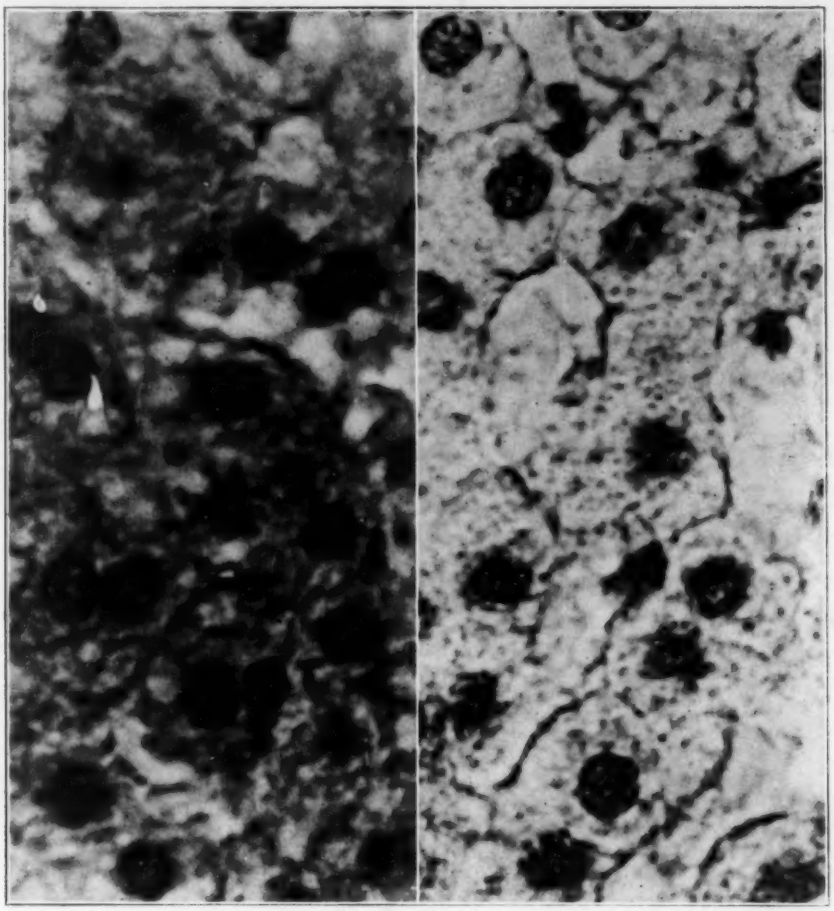

Fig. 7.—Effects of fright on the liver of a rabbit (reduced from photomicrographs, × 1,640): *A*, section of normal suprarenal of a rabbit; *B*, section of suprarenal of a rabbit after repeated periods of fright.

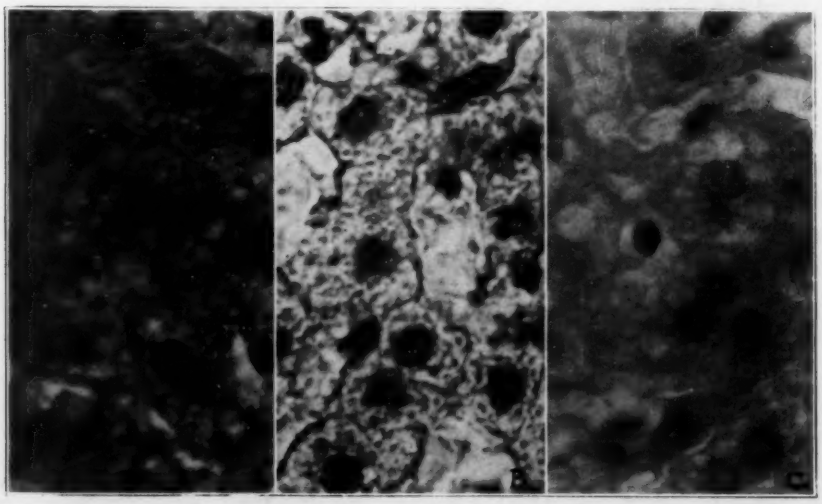

Fig. 8.—Immediate and late effects of fright on the livers of woodchucks (reduced from photomicrographs, × 1,640): *A*, section of normal liver of woodchuck; *B*, section of liver of woodchuck killed immediately after one period of fright, and, *C*, section of liver of woodchuck killed four hours after one period of fright.

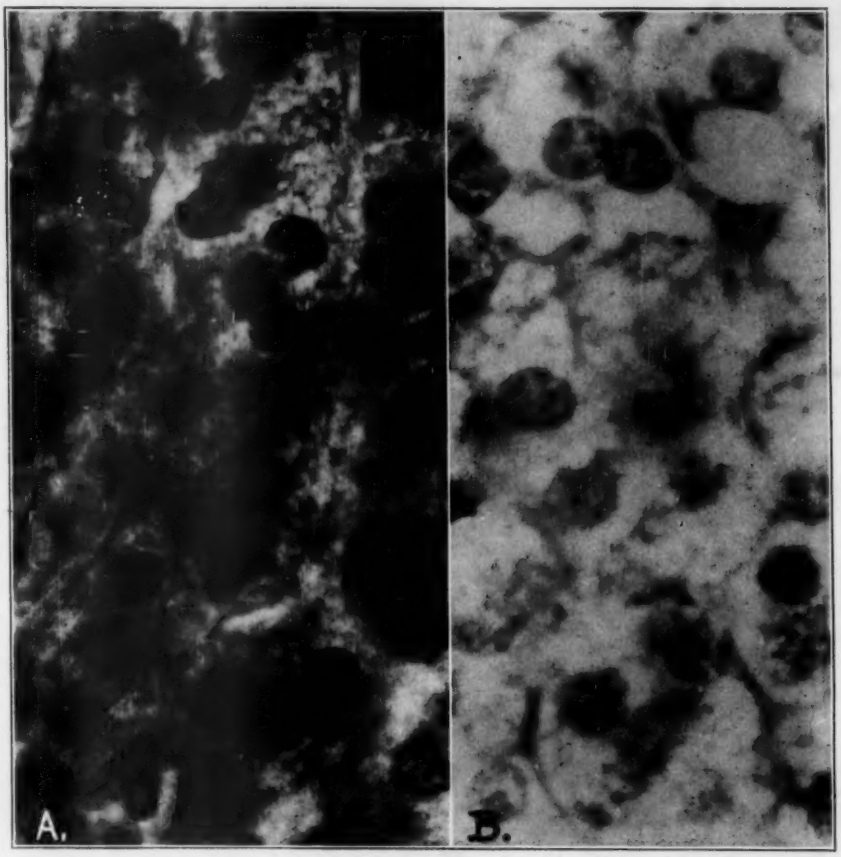

Fig. 9.—Effect of fright on the suprarenal of a rabbit (reduced from photomicrographs, × 1,640): *A*, section of normal suprarenal of a rabbit; *B*, section of suprarenal of a rabbit after repeated periods of fright.

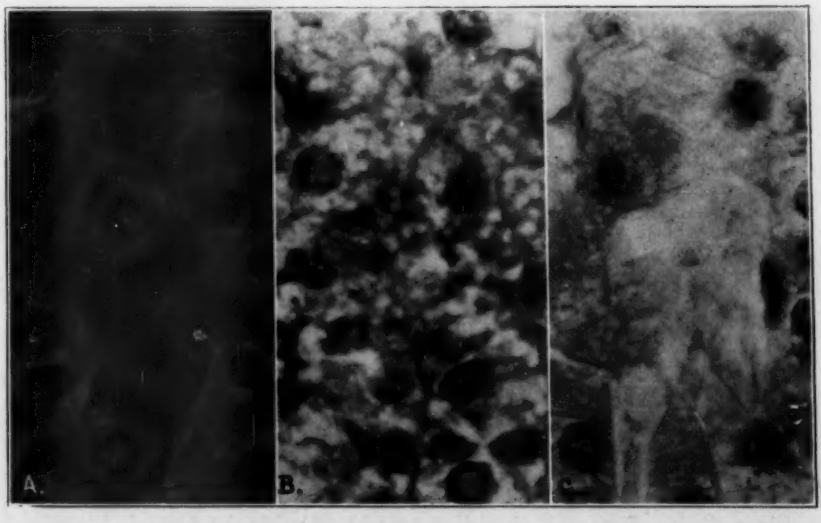

Fig. 10.—Immediate and late effects of fright on the suprarenals of woodchucks (reduced from photomicrographs. × 1,640): *A*, section of normal suprarenal of a woodchuck: *B*, section of suprarenal of a woodchuck killed immediately after a single period of fright, and *C*, section of suprarenal of a woodchuck killed four hours after a single period of fright.

TABLE 11.—Histologic Studies of Fear

Animal	Description of Experiment	Brain	Suprarenals	Liver
Rabbit	½ hour fright followed by subcutaneous injection of 4 c.c. per kg. 25% solution magnesium sulphate; killed in 6 hours	Cerebellum: only an occasional hyperchromatic and active cell; almost all fatigued; few exhausted Cerebrum: no hyperchromatic cells; many active and fatigued cells	Cortex: slight loss of cytoplasm and vacuolation in outer cells Medulla: pale with loss of cytoplasm in some cells	No loss of cytoplasm or vacuolation; stain not sharp
Rabbit	½ hour fright followed by subcutaneous injection of 4 c.c. per kg. 25% solution magnesium sulphate; killed 6 hours later	Cerebellum: occasional hyperchromatic and active cell; almost all fatigued; few exhausted Cerebrum; some hyperchromatic cells; some active; many fatigued; few exhausted	Cortex: loss of cytoplasm and vacuolation in outer cells Medulla: pale with loss of cytoplasm in some cells	Moderate uniform loss of cytoplasm in all cells
Cat	½ hour fright followed by 2 intravenous injections of 10 c.c. saturated solution Na_2HCO_3 ten minutes apart; killed 4 hours after second injection	Cerebellum: some hyperchromatic cells; most all active; some fatigued cells; stain intense	Cortex: marked loss of cytoplasm and vacuolation	Slight uniform loss of cytoplasm

TABLE 12.—Effect of Fright on the Suprarenal Output
(Cannon test)

Animal	Duration and Manner of Fright	Epinephrin Test 1	Epinephrin Test 2	Epinephrin Test 3
Female cat	Frightened 15 min. by bulldog	Before fright negative	Immediately after fright positive	10 min. after fright negative
Large male cat	Frightened 15 min. by bulldog	Before fright slightly positive reaction	Immediately after fright positive	15 min. after fright positive
Female cat	Frightened 15 min. by bulldog	.....	2 min. after fright slight reaction	
Male cat	Frightened 13 min. by bulldog; intense fright	Before fright negative	Immediately after fright positive	10 min. after fright slight reaction

by either Cannon's or Elliott's method[3] (Figs. 11 and 12). The protocols given in Table 12 give positive evidence that increased activity of the suprarenals is produced by fright, while the findings shown in Table 13 indicate that the stimulus which induces this increased activity is transmitted to the suprarenals from the brain (Fig. 13).

3. Elliott, T. R.: J. Physiol. **32**:401-406, 1905; ibid. **44**:374-409, 1912; ibid. **46**:285-290, 1913; ibid. **49**:38-53, 1914. Cannon, W. B.: Am. J. Physiol **33**:356-372, 1914. Cannon, W. B., and de la Paz, D.: Am. J. Physiol. **28**:64-70, 1911. Cannon, W. B., and Cattell, McK.: Am. J. Physiol. **41**:39-57, 58-72, 74-78, 1916. Hitchings, F. W., Sloan, H. G., and Austin, J. B.: Cleveland M. J. **12**:684-691, 1913. Stewart, G. N., and Rogoff, J. M.: J. Exper. Med. **24**:709 (Dec.) 1916; J. Pharm. and Exper. Therap. **8**:479-524, 1916.

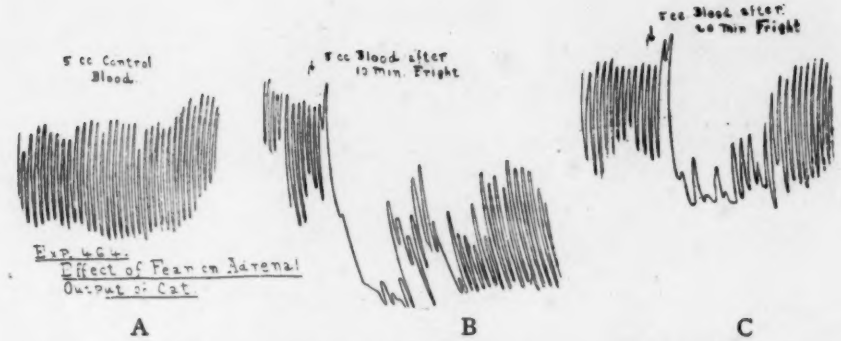

Fig. 11.—Tracing showing effect of fear on the suprarenal output of a cat (Cannon test). The first tracing *A*, was made by the contractions of intestinal muscle in blood from a normal cat. The contractions of the intestinal muscles were inhibited when the normal blood was replaced by blood from the same animal after it had been frightened. This inhibition is evidence of increased epinephrin in the blood stream, more epinephrin apparently is produced in the early stages of fright (*B*) than in the later stages (*C*).

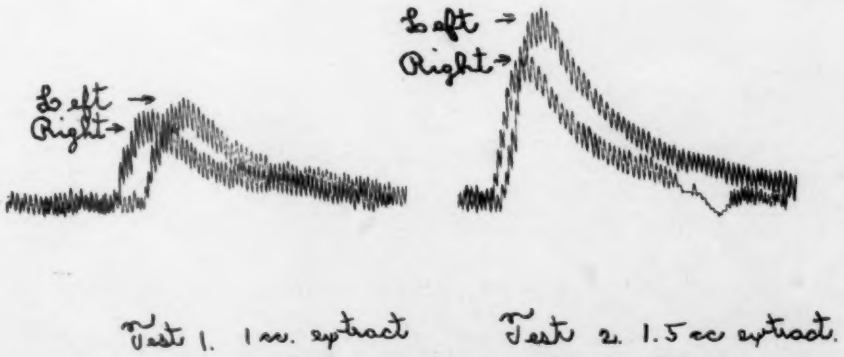

Fig. 12.—(Exp. 756, Jan. 16, 1914).—Tracing showing effect of fear on the epinephrin content (Elliott test). After the removal of the left gland, the animal (a cat) was frightened, after which the right gland was removed and the epinephrin content tested by the Elliott method.

TABLE 13.—Effect of Fright on the Suprarenal Output After Division of Splanchnics

(Cannon test)

Animal	Anesthetic and Operation	Duration and Manner of Fright	Epinephrin Test 1	Epinephrin Test 2	Epinephrin Test 3
Large male cat	N_2O plus local block abdominal wall; major splanchnics cut	Frightened 7 min. by bulldog	7 min. after fright negative	20 min. after fright negative	Epinephrin added to blood positive
Immature female cat	N_2O tracheotomy; 4 splanchnics cut above diaphragm	Frightened 5 min. by bulldog	Immediately after fright negative		
Female cat	N_2O tracheotomy; 4 splanchnics cut	Frightened 5 min. by bulldog	Negative		
Male cat	N_2O tracheotomy; splanchnics cut	Frightened 5 min. by bulldog	Negative		

TABLE 14.—Effect of Fear on the H-ion Concentration of the Blood

	P_H
Experiment 1.	
Normal blood from rabbit	7.58
Immediately after rabbit had been frightened by a fox terrier for 5 minutes	6.99, 6.91, 6.98
Ten minutes after termination of period of fright	7.19
Experiment 2.	
Normal blood from rabbit	7.65, 7.67
Immediately after rabbit, confined in box,* had been frightened by dog for 10 minutes	7.17, 7.17
After rabbit had been kept quiet in box for 45 minutes and then again frightened for 5 minutes	7.24, 7.41
After rabbit had rested 30 minutes	7.58, 7.60
Experiment 3.	
Blood from femoral vein of cat. Cat tied down and vein exposed under cocain	7.43
After cat had been frightened for 10 minutes by a dog	7.20
One hour later; animal struggling, respiration very rapid	7.37
Experiment 4.	
Normal blood from rabbit	7.62, 7.62
After rabbit had been frightened for 3 minutes by a fox terrier	7.54, 7.54
Fifteen minutes after the intravenous injection of 1.5 c.c. 2 per cent. morphin	7.52
Sixty minutes later	7.57
Immediately after administration of ether for 5 minutes	7.32, 7.32
Experiment 5.	
Blood from cat which had been tied down and was struggling	7.09, 7.14
Immediately after cat had been frightened for 15 minutes by a dog	7.16
One hour later after intravenous injection of 1½ grains (0.1 gm.) morphin sulphate in divided doses	6.99
Three hours after fright, cat dying	6.98
Experiment 6.	
Blood from the vena cava of a cat, taken through a catheter passed to the vena cava through the femoral	7.21, 7.24
Immediately after cat had been frightened by a dog for 15 minutes	7.02, 6.97
Immediately after the subcutaneous injection of 15 m. 1:1000 epinephrin chlorid	6.95, 6.95
Forty-five minutes later	6.89, 6.89

* By keeping rabbit in a box during the seances of fright the element of exertion was in part eliminated.

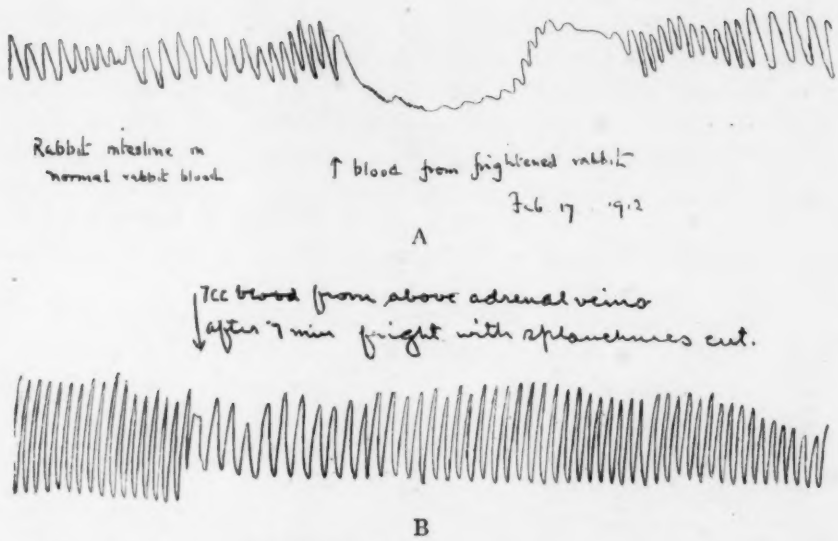
Rabbit milestone in normal rabbit blood
↑ blood from frightened rabbit
Feb 17 '13
A
7cc blood from above adrenal veins after 7 min fright with splanchnics cut.
B

Fig. 13.—(Exp. 460, Tracing I, March 6, 1913).—Comparison of the effect of fright on (*A*) the suprarenal output of a normal rabbit with the effect of fright on (*B*) the suprarenal output of a rabbit after division of the splanchnics. (Note the apparent lack of suprarenal response in *B*.)

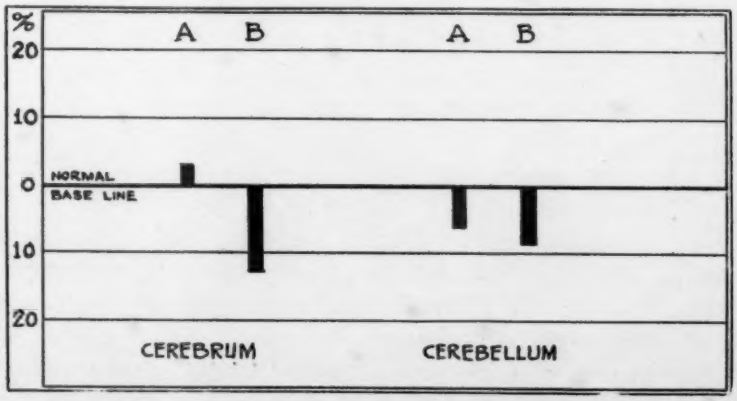

Fig. 14.—Changes in the electric conductivity in the brains of rabbits produced by fright (percental variations): *A*, after single seance of fright, and, *B*, after repeated seances of fright.

EFFECT OF FEAR ON THE H-ION CONCENTRATION OF THE BLOOD

Six studies of the effect of fright on the H-ion concentration of the blood, made by Dr. Menten,[4] showed that in every instance the H-ion concentration was markedly increased. This increased acidity was evidenced clinically by a greatly increased respiratory rate. The neutralization of the increased acidity was retarded in the two cases in which morphin was administered after the period of fright, and in one instance in which epinephrin was given.

In this series of experiments, no measurements of the H-ion concentration of the urine or of other body fluids were made (Table 14).

PHYSICOCHEMICAL STUDIES OF THE EFFECTS OF FEAR[5]

In order to determine the effect of fear on the chemical content of the thyroid, suprarenals, liver and muscle, ten rabbits were frightened

TABLE 15.—PHYSICOCHEMICAL STUDIES OF THE EFFECT OF FEAR ON CERTAIN ORGANS AND TISSUES

Series	Thyroid; Iodin Content	Suprarenals: Epinephrin Content, per Cent.	Suprarenals: Epinephrin Activity, per Cent.	Glycogen Content, per Cent.	Muscles; Glycogen Content
A. 12 normal rabbits	0.25 mg.	0.04	2	1.06	
12 rabbits after fright 1 hour	0.47 mg.	0.033	1.67	1.42, 0.745 (duplicate assay)	
B. 10 normal rabbits	1.11 mg. 0.04%	0.025	1.25	1.01	Trace
10 rabbits after fright ½ hour	2.48 mg. 0.19%	0.030	1.5	1.25	Trace

by dogs as in the experiments described above. The glands and tissues from these rabbits and also from an equal number of normal rabbits were sent to Parke, Davis and Company, where assays were made through the courtesy of Dr. Houghton, and Messrs. Biggs, Hamilton and Rome by the methods described in the preceding section of these studies in exhaustion.[1]

The results are given in Table 15. In each of these tests, the iodin content of the thyroid gland is increased; but the variation in the findings in the liver, suprarenals, and muscle indicates the need for many additional experiments.

In our own laboratory, we confirmed Cannon's findings of glycosuria in cats, but not in rabbits.

In five experiments in which the urine was tested prior to and after the seance of fright, albumin and casts appeared.

4. Menten, M. L., and Crile, G. W.: Am. J. Physiol. **38**:225-232, 1915.

5. Cannon, W. B., and Lamson, P. D.: J. Pharm. & Exper. Therap. **7**:167-171, 1916.

In four experiments, performed by Prof. R. D. Milner and Dr. C. A. Bowers, measurements of the basal metabolism were made after seances of fright, and compared with the basal metabolism of a series of normal rabbits. The results are shown in Table 16.

TABLE 16.—CHANGES IN THE METABOLISM OF RABBITS CAUSED BY FEAR

	Weight of Animal, Gm.	Time, Minutes	Corrected Measurements, C.c. CO_2	Corrected Measurements, C.c. O_2	R. Q.	Remarks
A. Normal Rabbits	2,304	60	1.003	1,247	0.80	Without food for two days; rabbit restless
		60	0.9571	1.126	0.85	
	2,334	40	0.7229	0.894	0.81	Fed after 4 p. m. preceding day; food allowed to remain in cage all night
		51	0.7993	1.005	0.80	
		64	0.9571	1.171	0.82	
	2,340	31	0.5397	0.636	0.85	Generous feeding after 4:30 preceding day; stomach full this morning
		60	1.059	1.168	0.90	
		60	1.018	1.134	0.90	
	2,346	30	0.5804	0.690	0.84	Fed on morning of preceding day
		30	0.5856	0.709	0.83	
		30	0.6618	0.768	0.86	
	2,346	45	0.7025	0.942	0.74	Fed previous afternoon
		45	0.7535	0.924	0.82	
		45	0.7891	0.928	0.85	
B. Frightened Rabbits	.....	45	0.8095	1.048	0.772	Rabbit frightened by dog until exhausted
		45	0.6873	0.863	0.796	
		45	0.6516	0.839	0.776	
	1,372	45	0.4378	0.594	0.74	Frightened by dog for 20 min.; very little apparent disturbance
		45	0.4684	0.618	0.76	
		60	0.6516	0.794	0.82	
	2,229	45	0.6415	0.997	0.64	Frightened by dog for one half hour
		45	0.7178	0.938	0.765	
		45	0.6211	0.787	0.789	
	2,219	45	0.8604	0.927	0.93	Frightened and chased by dog for 25 min. until completely exhausted
		45	0.6771	0.963	0.70	
		45	0.6669	1.010	0.66	

TABLE 17.—AVERAGE SPECIFIC CONDUCTIVITY OF THE BRAINS OF NORMAL RABBITS
(expressed in reciprocal ohms)

	Number of Animals	Average Deviation	Percental Variation	Average Conductivity
Cerebrum	6	2.7	1.4	0.00189
Cerebellum	6	1.4	0.8	0.00164

CHANGES IN THE SPECIFIC ELECTRIC CONDUCTIVITY OF THE BRAIN PRODUCED BY FEAR

Electric conductivity measurements of various tissues of Belgian hares were made by Miss H. R. Hosmer and Miss A. F. Rowland after single and after repeated seances of fright, conducted as nearly as possible in an identical manner with the experiments in the previous phases of this research. Both the normal and the frightened animals in this group were purchased from the same dealer, and had been kept for some time under identical conditions in our laboratory. The measurements from these normals and from the frightened animals are given in Tables 17 and 18, and the results on the conductivity of the brain are shown graphically in Figure 14.

CLINICAL EFFECTS OF FEAR

Temperature.—In most of our experiments the temperature rose, sometimes as much as 3 degrees Centigrade, the maximum usually being reached in from fifteen to twenty minutes. Marinesco has shown that a much higher temperature than this is required to cause brain cell changes from heat alone. In two rabbits in which complete thyroidectomy had been performed two weeks before the experiments, the temperature *fell* instead of rising; in one case 2 degrees and in the other 5 degrees Centigrade.

TABLE 18.—SPECIFIC CONDUCTIVITY OF THE BRAINS OF RABBITS AFTER SINGLE AND AFTER REPEATED SEANCES OF FRIGHT
(expressed in reciprocal ohms)

		Cerebrum	Cerebellum
After single seance of fright	Rabbit 1	00199	00144
	Rabbit 2	00191	00156
	Rabbit 3	00195	00163
Average		00195	00154
After repeated seances of fright	Rabbit 1	00157	00153
	Rabbit 2	00161	00147
	Rabbit 3	00178	00149
Average		00165	00150
Normal average		00189	00164

TABLE 19.—EFFECT OF FEAR—REPEATED TEN MINUTE PERIODS—ON THE TEMPERATURE OF RABBITS

	Group I						Group II			
	Rabbit 1		Rabbit 2		Rabbit 3		Rabbit 1		Rabbit 2	
Day	Before Fright	After Fright	Before Fright	After Fright	Before Fright	After Fright	Before Fright	After Fright	Before Fright	After Fright
1	39	39.5	37	39	37	40	39.25	39.5	39.25	39.75
2	39	40	37.5	40	37	40	39	39.5	39.25	39.25
3	37	39	37.5	39.5	39	39	37.5	39.75	38.75	
4	37	39	.....	.....	38.25	39.5	39	39.5	38.5	38.75
5	37	38.5	.....	.....	37	38	39.75	41	40	40.75
6	37	38.5	.....	.....	37	38.5	39.5	39.25	39	40.5
7	37	38.75	.....	.....	37.5	40	39.25	39.5	39.5	39.5
8	36	36.25	.....	.....	37	37.5	39	40	39	39.5
9	37.5	.....	.....	.....	37	.....	39.25	40	39	39.75
10	38.5	39.25	.....	.....	38.5	39.25	39.5	39.75	39.5	39.75
11	38.5	39.75	.....	.....	39	39.25	38	39	38	38
12	38.25	39.25	.....	.....	39	39				
13	39	41	.....	.....	38	41				
Average elevation of temperature					1.3 C. Rabbit 1 2.2 C. Rabbit 2 1.48 C. Rabbit 3		Average elevation of temperature		0.7 C. Rabbit 1 0.45 C. Rabbit 2	

In two experiments in each of which rabbits were frightened for ten minutes twice daily for several successive days, the temperature changed during each seance of fright as shown in Table 19 (temperature given in degrees Centigrade).

Respiration.—The respiratory rate was always increased—in one instance to 140 per minute.

Pulse.—The heart beat was accelerated.

Blood Pressure.—The average blood pressure of seven normal rabbits was 76 mm. of mercury; that of two frightened rabbits was 93 mm. of mercury.

In all the experiments, half an hour of continued fright exhausted the rabbit completely, as was evidenced by the gross clinical phenomena and the position—flat on the floor with head and legs extended—assumed as soon as the cause of fear was removed.

HUMAN PHENOMENA

In persons under emotional stress the increased activation is manifested by the facial expression and by muscular and mental phenomena. The increased suprarenal activity is evidenced by the inhibition of the intestines; the participation of the liver, by glycosuria; the muscular factor, by the trembling inhibition of movement, muscular fatigue, and increased temperature; the increased H-ion concentration preeminently by the increased respiratory rate. If the emotional stress continues over a prolonged period, the thyroid gives evidence of its participation by enlargement and by the phenomena, which if they become established are characteristic of the condition designated as exophthalmic goiter.

The phenomena of this disease present striking evidence of the participation of each organ in the kinetic chain in emotional response.

During the World War, the human phenomena of intense activation, in which activation due to the emotions played a predominant rôle, were manifested on a vast scale—in the noncombatants at home, in the refugees, in the soldiers in the field and in hospitals, and in the prisoners of war. As the tide of war approached and engulfed each individual, his resultant activation was expressed in intensified consciousness—in insomnia, in exertion and in emotion, to the results of which, according to the fortunes of the individual, the stimuli of physical injury and infection may have been added.

At the American Ambulance in January, 1915, we were able to secure specimens of the brains, livers and suprarenals of soldiers who had participated in the great retreat of the allied armies from Mons to the Marne. During this retreat, goaded by shot and shell and the ever advancing enemy, they had marched for nine days and nights without adequate sleep and food, and in constant fear of capture. Each had been wounded, the wounds of each had been infected to some degree; but in no instance was either wound or infection sufficient to cause death. In the fatal exhaustion of these men, it is obviously impossible to assign the predominant rôle to insomnia, to exertion or to emotion—each unquestionably contributed its share to the fatal result. The results of the histologic examination of the brains, livers and

TABLE 20.—Results of Histologic Examinations in Soldiers

	Cerebellum	Cerebrum	Medulla	Suprarenal	Liver
Soldier 1	No hyperchromatic cells; very few active, many exhausted, nearly all fatigued	All fatigued and exhausted	No hyperchromatic cells; few active; nearly all cells fatigued with marked loss of cytoplasm and eccentric nuclei; cytoplasm very finely granular	Outer two thirds of cortex shows loss of cytoplasm and moderate vacuolation; glomerular layer not so much affected as the central part of the cortex	Slight loss of chromatin and small vacuoles in most cells except in periphery of lobules where there is large vacuolated appearance
Soldier 2	No hyperchromatic cells; few active; nearly all badly fatigued; remaining cytoplasm in cells very finely divided; many exhausted cells	No hyperchromatic or active cells; all fatigued or exhausted, cytoplasm very diffuse	Olive cells all fatigued with marked loss of cytoplasm	Slight loss of chromatin and vacuolation of outer cells of cortex	No loss of cytoplasm but granules not distinct; no vacuolation
Soldier 3	No hyperchromatic or active cells, all badly fatigued or exhausted; loss of cytoplasm almost complete with exception of nucleoli	No hyperchromatic cells but many active cells with diffuse chromatin; no distinct granules; not many exhausted cells	Olivary cells slightly fatigued; motor cells fatigued with many eccentric nuclei	Very slight loss of cytoplasm of an occasional cell; nearly all normal	No loss of cytoplasm or vacuolation; granular degeneration of cytoplasm
Soldier 4	No hyperchromatic or active cells; all cells fatigued or exhausted	No hyperchromatic or active cells; nearly all fatigued; some exhausted	Cells in various stages of fatigue; many active olivary cells; some fatigued	Slight loss of cytoplasm of cortical cells	No loss of cytoplasm or vacuolation; stain weak and diffuse
Soldier 5	Occasional active cell; nearly all fatigued	Some hyperchromatic cells; many active; occasional cell fatigued	Occasional active cell; nearly all fatigued	No loss of cytoplasm or vacuolation	No loss of cytoplasm or vacuolation

suprarenals of these soldiers are therefore included here, since they illustrate the effects of the acme of intensified consciousness due to the utmost exaggeration of normal activations (Table 20 and Figs. 15 and 16).

CONCLUSIONS

1. Extreme emotion causes demonstrable histologic lesions in the central nervous system, the liver and the suprarenals. The first effect of fear, as evidenced by the histologic changes in the brain cells, is increased activity, manifested by hyperchromatism followed by a progressive chromatolysis if the activation is continued. After a single seance of intense fear, the initial hyperchromatism is apparently followed by a period of hypochromatism with a gradual return to the normal.

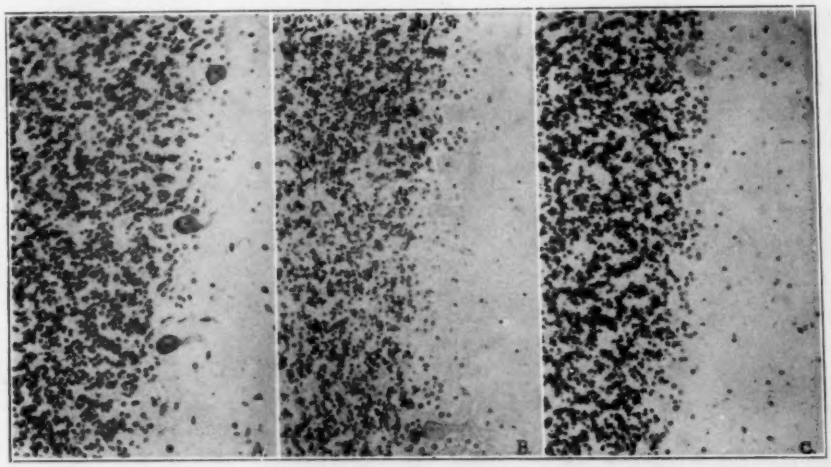

Fig. 15.—Effect of exhaustion from insomnia, exertion and emotion on the brains of soldiers (reduced from photomicrographs, × 310): *A*, section of normal human cerebellum; *B* and *C*, sections of cerebellum of exhausted soldiers. Note the disappearance of Purkinje cells.

2. Fear causes glycosuria (in cats), albumin and casts in the urine, diminished metabolism, and changes in the iodin content of the thyroid.

3. The hydrogen-ion concentration of the blood is increased by fear.

4. Preliminary observations of the changes in the specific conductivity of the cerebrum produced by fear indicate that they parallel the histologic changes; i.e., there is first an increased conductivity followed by a decrease to below the normal.

5. Clinically, extreme emotion raises the body temperature, increases the respiratory and pulse rate; and, if sufficiently intense and prolonged, produces a complete prostration which may prove fatal.

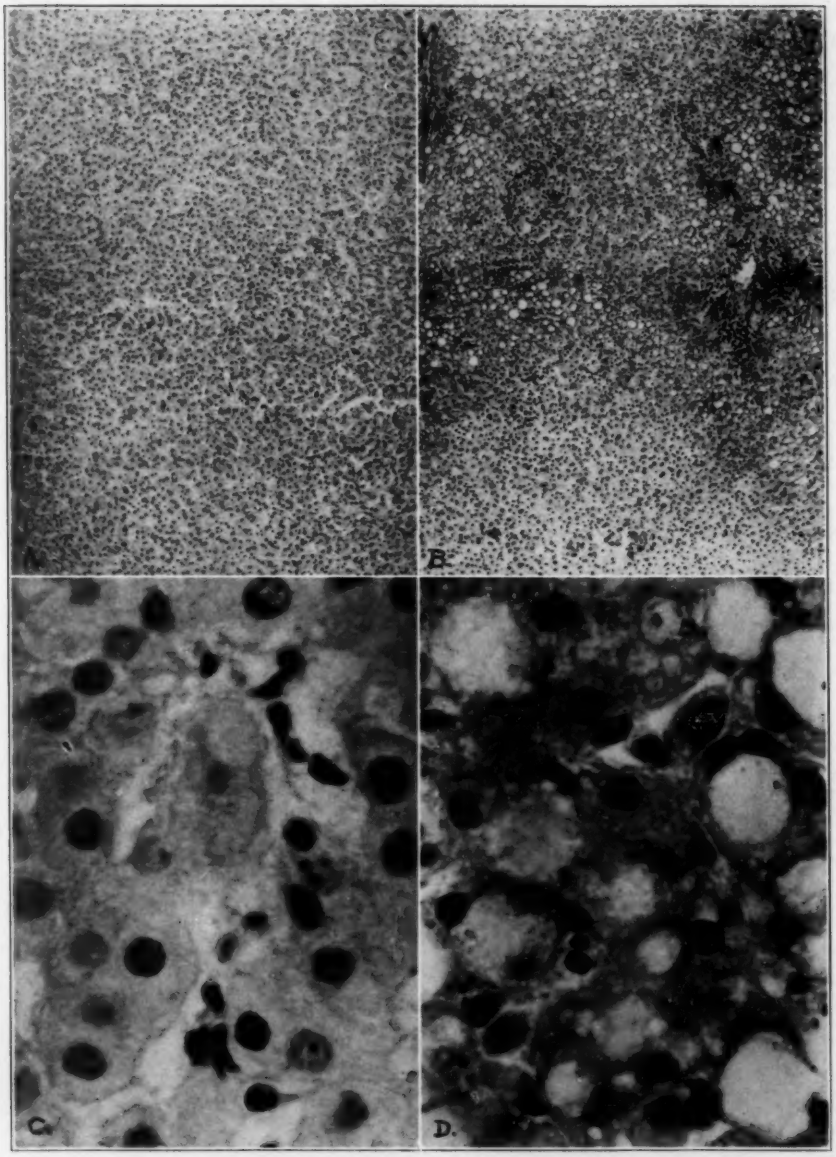

Fig. 16.—Effect of exhaustion from insomnia, exertion and emotion on the livers of soldiers: *A* and *C*, sections of normal human liver; *B* and *D*, sections from the livers of exhausted soldiers. In *B* and *D* the disappearance of cytoplasm and vacuolated spaces as compared with the evenly distributed cytoplasm in *A* and C may be noted. (*A* and *B* from photomicrographs, × 120; *C* and *D* from photomicrographs, × 1,640.)

SUMMARY

The emotive response of timorous animals is a commonplace. As a human experience, it is universal. That it may be graded in intensity up to a critical point is acknowledged; that it may be overwhelming and suspend function is commonly observed. In our researches, we used many animals, and found, as Colonel Mott [7] has concluded, that the emotive response is one of the most powerful of which the organism is capable. Emotion causes a more rapid exhaustion than is caused by exertion, or by trauma, excepting extensive mangling of tissue, or by any toxic stimulus except the perforation of viscera. Apparently, in birds, in particular, the emotion of fear may instantly overwhelm the organism, as when a bird is unexpectedly confronted by a snake.

In our experiments, fear caused profound changes in the cells of the brain, the liver, and the suprarenals; in some cases an acidosis developed acutely; in some cases albumin and sugar appeared in the urine; the epinephrin output, as has been demonstrated by Cannon, was increased; the electric conductivity of the brain, the liver and of other organs was altered.

In short, our researches have shown that the emotions drive the organism with extreme intensity; that, like trauma or exertion, emotion may drive the organism within the limits of normal response, or so overwhelmingly as to suspend the normal functions and reduce the individual to a state of complete, cold prostration. In other words, emotion may cause exhaustion; it may cause shock.

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7. Mott, F. W.: War Neuroses and Shell Shock, New York, Oxford University Press, 1919.

STRUCTURAL RESULTS OF PROSTATECTOMY WITH REFERENCE TO METHODS OF ENUCLEATION

BASED ON A STUDY OF ONE HUNDRED AND THIRTY-SIX CASES *

FRANK HINMAN, A.B., M.D.

SAN FRANCISCO

A recent analysis of my own cases of prostatectomy [1] shows a close relationship between functional and structural results and emphasizes the fact that good anatomic restoration is essential to a complete cure of the prostatism. The failure of good posterior urethral restoration and of preservation of sphincters and ejaculatory ducts may be the result of anatomic changes produced by the disease or by the surgical procedure. It is seldom that uncomplicated prostatism can so alter anatomic structures as to be incurable. Unsuccessful and incomplete enucleation of the hyperplastic mass is, however, one of the chief surgical errors that lead to immediate functional failure, or later, to recurrence of prostatism. A knowledge of the pathology of prostatic enlargement, with the anatomic changes that accompany it, and a careful consideration of these facts in relation to either the suprapubic or perineal method of enucleation are essential to an understanding of the structural defects peculiar, as well as common, to both. Such an understanding, if well founded, should reduce the frequency and gravity of structural defects surgically produced and lead in consequence to marked improvement in functional results.

PATHOLOGY AND ANATOMIC CHANGES

The facts regarding the anatomy and pathology may be briefly stated. Hyperplasia begins in the central group of glands in separate foci and probably never as a diffuse process. Numerous spheroidal tumors result, which tend to agglomerate, and in their growth condense the peripheral gland tissue by compression to form a pseudocapsule. It is inside this false capsule that surgical enucleation is performed.[2] The posterior lobe is separated from the other lobes by

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*Read before the Section on Urology at the Seventy-Second Annual Session of the American Medical Association, Boston, June, 1921.

1. Hinman, Frank: "Suprapubic versus Perineal Prostatectomy, a Comparative Study of Ninety Perineal and Thirty-Eight Suprapubic Cases," read before the American Urological Association, Montreal, June 2, 1921.

2. Freyer ("Clinical Lectures on Enlargement of the Prostate with a Description of the Author's Method of Total Enucleation of the Organ," William Ward Company, 1920) contends that he performs a complete pros-

the connective tissue sheath carrying the ejaculatory ducts and apparently does not undergo hyperplastic changes, so that for all practical purposes all enlargements are prespermatic. Any peri-urethral group of glandular tissue of the middle, lateral or anterior lobes may be the initial focus of hyperplasia.[3] These changes may also occur in the subcervical and subtrigonal groups and cause obstruction; but it is, indeed, rarely, if ever, that one sees enlargement of these groups without an associated enlargement of some of the chief lobes of the prostate. The type of enlargement will necessarily conform to the number and location of initial foci and their relative growth. All sorts of combinations have been observed; but the commonest is bilateral and middle lobe enlargement which is peri-urethral and more or less fused into one mass. The histopathology of this mass varies from an almost pure adenoma to myoma or fibroma; but most frequently it is a mixed fibromyo-adenoma with the glandular element predominating. The more fibrous or muscular types are smaller, the former commonly complicated by or secondary to prostatitis, and both likely to be unassociated with a false capsule. An enlargement which has no false capsule or line of cleavage is recognized as difficult of enucleation. Growth

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tatectomy. He bases his conclusion on the following evidence (p. 156): "(1) The general conformation of the specimens removed by me indicated that they were entire prostates. (2) The absence of any palpable substance in the cavity that remained at the time of operation, as felt between a finger in this cavity and a finger in the rectum, a very thin membrane consisting merely of the bowel and sheath of rectovesical fascia lying between the points of the fingers. (3) The absence of any mass resembling prostatic tissue in the cases operated on, at any period after the operation, on examination by the finger in the rectum. (4) When the enlarged prostate projects prominently in the bladder the true capsule is at once reached on scraping through the mucous membrane covering it by the finger nail. (5) The absolute and complete relief of symptoms after operation. (6) But Mr. Thompson Walker has adduced the most cogent evidence of all by demonstrating that no prostatic tissue is found in specimens removed from the bodies of persons on whom the operation had been performed during life." It is rather hazardous to disagree with two such eminent authorities, nevertheless, I do not hesitate to do so. For all practical purposes, complete enucleation of the prespermatic portion in cases of large glands is effected by the Fuller-Freyer as well as by the radical *en masse* perineal method. The false capsule of compressed nonhyperplastic gland tissue is insignificant in these cases. In smaller enlargements, however, there is a very visible and evident false capsule composed of more or less normal gland tissue. This can be clearly demonstrated in postmortem examinations of unoperated cases of prostatism. A complete enucleation would remove the vermontanum and terminal portions of the ejaculatory ducts. This, I believe, is often done suprapubically, as has been demonstrated by a number of our perineal specimens before we appreciated the importance of incision of the supramontane urethra.

3. Freyer conceives the prostate as formed of two lateral lobes.

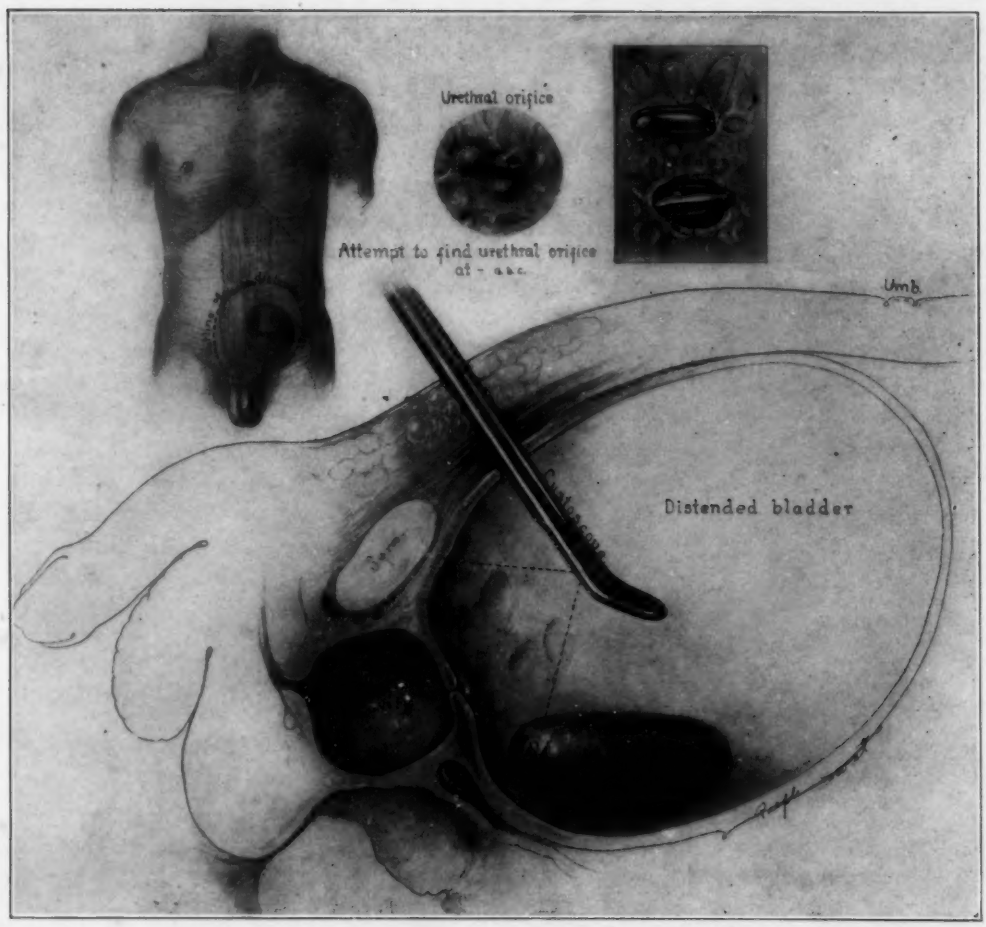

Fig. 1.—Diagrammatic representation of conditions as found in a man, aged 74, five years after suprapubic prostatectomy. He had never voided through the urethra since operation and had worn a suprapubic tube for five years. Repeated previous attempts to catheterize or dilate the urethra were unsuccessful. There were marked pyuria and renal insufficiency; a very large abdominal hernia through the scar of the suprapubic incision. There was no suspicion or evidence of cancer. Cystogram revealed double cavity, and it was at first thought that we were dealing with a diverticulum. Cystoscopically a very large prostatic cavity with an orifice that closely simulated the mouth of a diverticulum was found, through which the shaft of the instrument could be passed with difficulty. The bladder was markedly dilated and contained a large stone. Passing the cystoscope through the suprapubic sinus, a lithotrite could be made to engage at the contracted vesical orifice through the urethra (as shown in inset) and the stone was crushed under cystoscopic control. Young's punch instrument was inserted in the same way and the vesical neck contracture enlarged (inset *a* and *b*).

follows the line of least resistance intravesically inside the internal sphincter, and with large glands, the internal sphincter may be much dilated. The supramontane portion of the urethra is elongated and distorted in accordance with the type of enlargement. The verumontanum and ejaculatory ducts are pressed posteriorly, and the inframontane portion of the prostatic urethra with the external sphincter is undisturbed. What problems with reference to the enucleation of this hyperplastic mass from its prostatic bed do these facts present to the surgeon?

Fig. 2.—Urethroscopic appearance of prostatic urethra four years after suprapubic prostatectomy; large granular and nodular cavity with dilated vesical orifice.

METHODS OF ENUCLEATION

Suprapubic enucleation is commonly a one-piece removal performed in one of two ways: Intra-urethrally or extra-urethrally; but the hyperplastic mass is sometimes removed in separate pieces either way. Intra-urethral *en masse* enucleation has been performed by us by forcing a finger back into the urethra and tearing it anteriorly. By blunt dissection on this line of cleavage, the hyperplastic mass is freed from within outward. In a few cases, we have made an annular or linear incision of the mucosa over the intravesical mass as an entering wedge for extra-urethral enucleation, from without inward, outside the urethra.

The extra-urethral would seem to be a method better suited to surgery, although more difficult, and probably more often secures a complete removal. Whenever the gland is not brought away in one piece, it is sometimes difficult to determine whether spheroids remain, even with bimanual palpation with the finger in the rectum, which few surgeons practice because of danger of sepsis. Any of these procedures must be performed through the internal sphincter, which is thereby dilated and sometimes destroyed. The supramontane portion of the prostatic urethra is torn or more commonly completely removed. Tags and flaps

Fig. 3.—Urethroscopic appearance of prostatic urethra two years after perineal prostatectomy; persistent nodule at right side of vesical orifice; tags in prostatic urethra and small sinus persisting at site of perineal fistula which has been permanently closed. The only functional disturbance is nycturia.

of mucosa are generally left at the prostatic margin or cavity. The prostatic cavity is packed for hemostasis, thus tending to perpetuate posterior urethral elongation and cavity formation.

Perineal enucleation, if Young's technic is followed, preserves the prostatic urethra throughout, and rarely is either the internal sphincter or vesical mucosa disturbed. The bilateral posterior lobe incisions render possible good preservation of the ejaculatory ducts and verumontanum. When properly performed, the external sphincter remains untouched. The gland is enucleated in separate portions, first the right,

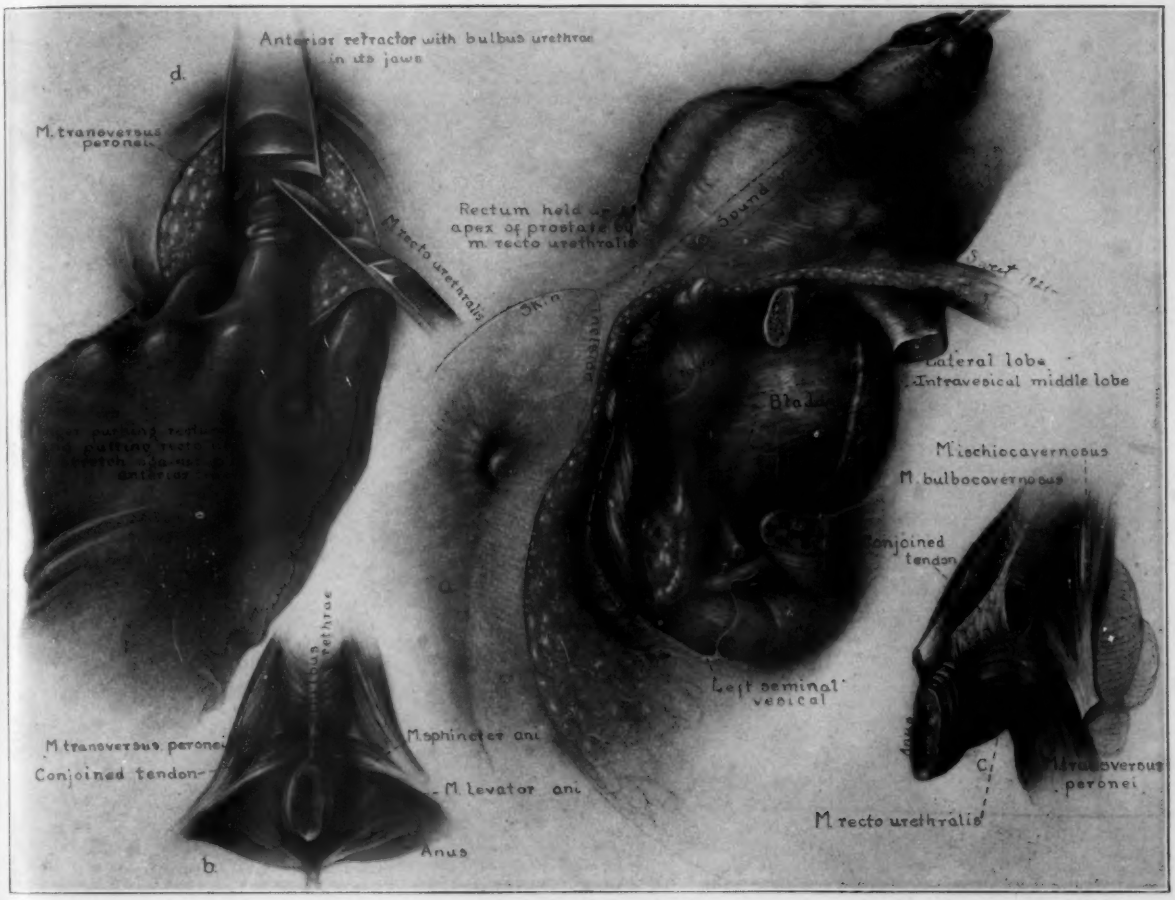

Fig. 4.—The all important recto-urethroprostatic juncture; *a*, close apposition of bulbus urethrae, triangular ligaments carrying the external sphincter, rectum and apex of the prostate; *b*, exposure of the membranous urethra keeping close to the rectum in order to preserve the perineal union and not injure the bulb, transversus perinei or triangular ligaments with the external sphincter; *c*, correct division of the conjoined tendon and recto-urethralis muscle; careless dissection may injure rectum or perineal union; *d*, separation of rectum from apex of prostate is best effected by knife and blunt dissection putting the recto-urethralis muscle on stretch by pressing rectum down with finger and pulling the bulb with the external sphincter and triangular ligaments up with anterior retractor.

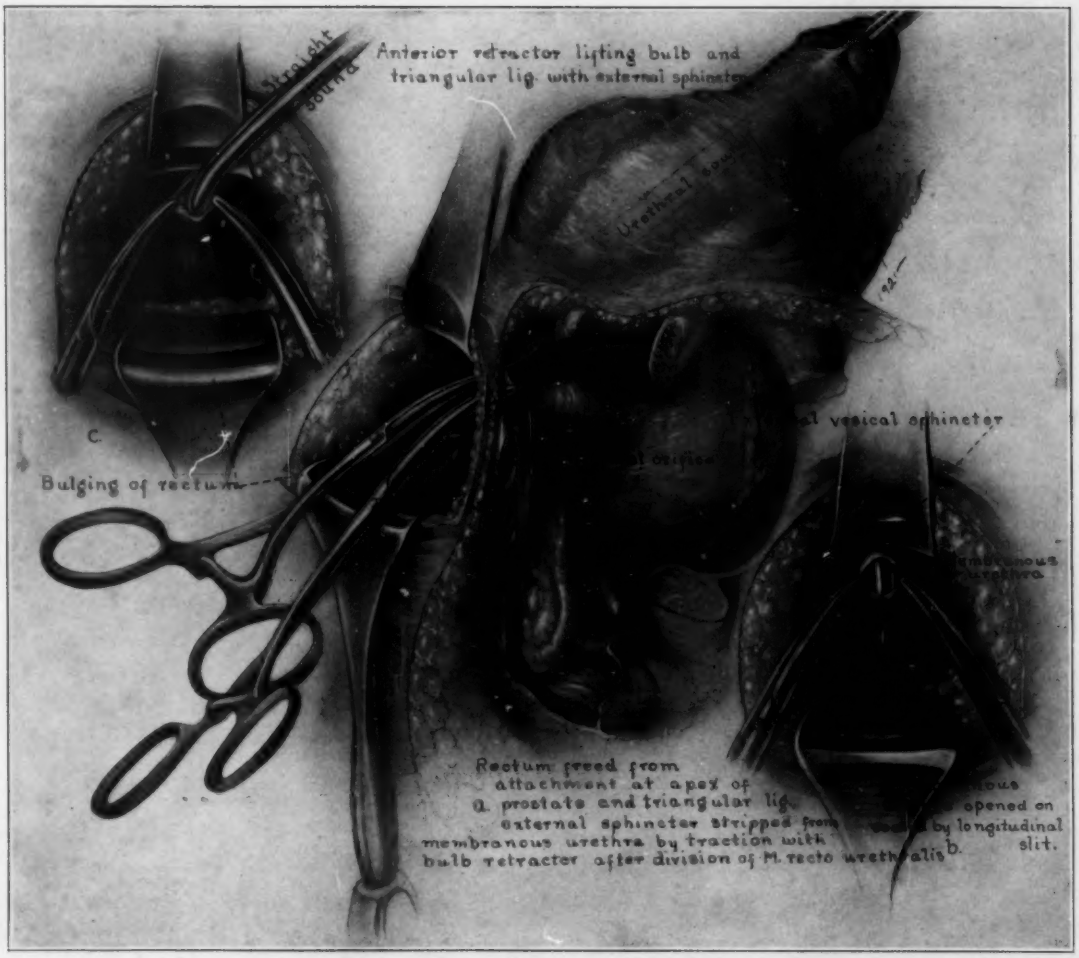
Straight sound
Anterior retractor lifting bulb and triangular lig. with external sphincter
Urethral sound
vesical sphincter
C.
Bulging of rectum
Rectum freed from attachment at apex of a. prostate and triangular lig.
external sphincter stripped from membranous urethra by traction with bulb retractor after division of M. recto urethralis
opened on by longitudinal slit.
b.

Fig. 5.—Exposure of the membranous urethra; *a*, clamps placed on each lip of the longitudinal incision; the rectum still in close contact with the apex of the prostate (*b*); *c*, the urethral sound removed by an assistant and the straight sound inserted into the bladder through the incision, acting as a control and guide to the insertion of Young's prostatic tractor.

then the left lobe, or vice versa, and an anterior or middle enlargement is finally delivered through one or the other of the lateral incisions. Successfully executed, the method is surgically ideal. Technical difficulties are increased with the ring type, and very large hyperplasias and these difficulties, probably more than the anatomic difficulties of approach, appeal to me as the important factors leading to the general disrepute of a perfect surgical procedure.

STRUCTURAL DEFECTS OF SUPRAPUBIC AND PERINEAL PROSTATECTOMY

The common defect in structure following suprapubic enucleation is a failure of restoration of the prostatic urethra, which is replaced by a larger or smaller cavity, either infundibular or spindle shaped, according to its association with a widely dilated or contracted vesical neck (Figs. 1 and 2). Incomplete enucleation may leave nodules or large lobes at various parts of the cavity or neck, which may be so placed as to impair sphincteric action, resulting in dribbling, or, to prevent complete evacuation, resulting in continued frequency and urinary infection. Tags of mucous membrane or other tissue left in the prostatic ensemble or about the neck may repair in such a way as to act as an irritant or even a ball valve polyp, producing marked disturbances of urination.

Cavity formation rarely follows the perineal operation; but structural defects at the neck from the presence of nodules or tags are common, and unless the operator is familiar with the perineal method, nodular remnants, due to incomplete removal of all the hyperplastic mass, are more likely to remain than after the suprapubic operation (Fig. 3). These cause functional disturbances and may lead to recurrence of prostatism. Deep fissure or sulcus formation is also not uncommon, particularly in cases after removal of very large glands, and, in such cases also, mucosal plication may lead to obstruction to instrumentation as well as urination.

MODIFIED METHOD OF PERINEAL ENUCLEATION

The radical *en masse* enucleation as usually done suprapubically insures more certain and complete removal of the hyperplastic tissue than does the lobular removal by the perineal method. This fact raises the question as to whether block enucleation through the perineum might not secure cleaner and more complete removal and at the same time give better structural restoration, or whether a one-piece perineal enucleation with divulsion of the prostatic urethra might leave, as is common suprapubically, a large prostatic cavity, or finally whether such radical removal would be more likely to injure the ejaculatory

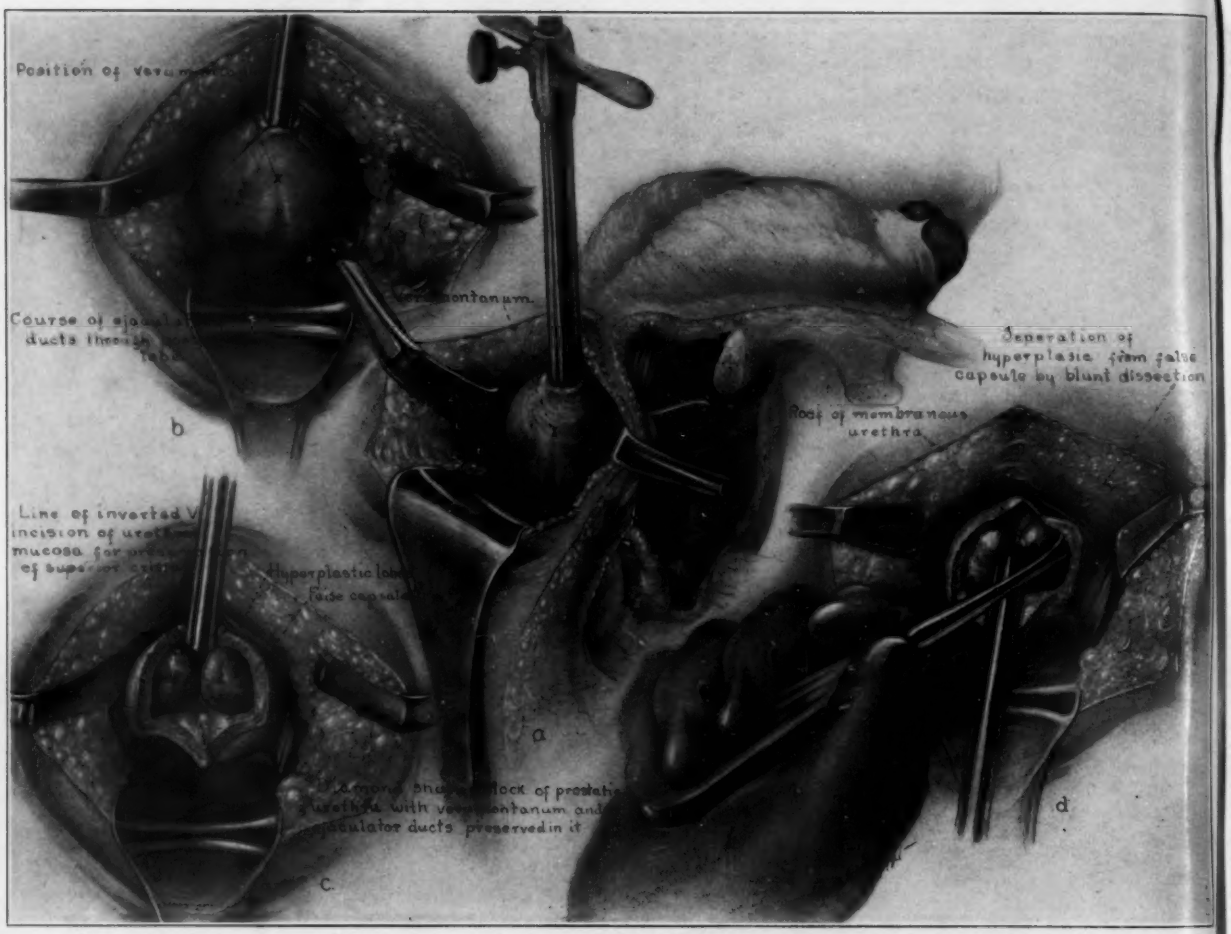
Position of verumontanum
Course of ejaculatory ducts through prostatic tissue
b
Line of inverted V incision of urethral mucosa for preservation of superior crista
Hyperplastic lobe
False capsule
a
Diamond shaped block of prostatic urethra with verumontanum and ejaculator ducts preserved in it
c
Separation of hyperplastic from false capsule by blunt dissection
Roof of membranous urethra
d

Fig. 6.—Method of enucleation: *a*, the rectum has been dissected from the posterior surface of the prostate along line of fascia of Dénonvillier and is held out of the way by the deep posterior retractor; the prostatic mass is drawn down into the wound by the prostatic tractor, and ready for incision for enucleation; *b*, a wide inverted V incision is made in the posterior lobe well into the hyperplastic mass; *c*, the pointed flap of posterior lobe when thrown back exposes the verumontanum and ejaculatory duct orifices. A diamond shaped block of prostatic urethra is preserved by a second inverted V incision of the mucosa with its apex just above the superior crista of the verumontanum; *d*, depression of the prostatic tractor exposes the roof of the membranous urethra which is divided with scissors so as to free completely the urethral end of the prostatic urethra. The hyperplastic mass is freed *en masse* by blunt dissection, or adhesions can be accurately divided with knife or scissors.

ducts. In order to arrive at an answer, we have now performed twenty-five perineal operations by a modification of Young's method of enucleation, whereby the hyperplastic gland may be removed in one piece just as is done suprapubically. The one purpose is to secure complete and clean removal and at the same time preserve the ejaculatory ducts, the verumontanum and both sphincters as effectively as by the old method. No attempt is made to preserve the supramontane portion of the urethra.

The technic of conservative perineal prostatectomy as practiced and taught by Hugh H. Young is followed up to the incision of the posterior lobe for the purpose of enucleation. The anatomic difficulties of approach of this operation are mastered with a thorough knowledge of the recto-urethro-prostatic juncture (Fig. 4). Injuries of the rectum or external sphincter never follow a properly performed perineal exposure.[4] The urethra at its membranous portion is opened by a longitudinal incision (Fig. 5), and after the insertion of the prostatic tractor, the rectum can easily be separated from the prostate along the line of Dénonvillier's fascia (Fig. 6*a*). After the prostate is well exposed, instead of making two lateral incisions into it, after Young's technic and as was done in our first sixty-five cases, an inverted V incision having a wide obtuse angle, the apex of which is at the opening into the membranous urethra, is used (Fig. 6*b*). This incision is deepened well into the lateral spheroids, or, if no lateral hyperplasia exists, is continued down through the urethral mucosa to the prostatic tractor (Fig. 6*c*). When so extended, it allows one to drop back the verumontanum and ejaculatory ducts in a triangular flap of the posterior lobe, thus giving wide exposure of the prostatic urethra (Fig. 6*c*). It is important now that the prostatic urethra above the verumontanum near the bladder be incised by another inverted V or elliptic cut so as to free the supramontane urethra completely from this flap portion (Fig. 6*c*), as otherwise the colliculus might be torn or mutilated by the manipulation to follow. Division of the roof of the membranous urethra with scissors (Fig. 6*d*) now frees the whole prespermatic portion. The hyperplastic mass can be easily and accurately dissected loose with perfect preservation of the internal sphincter (Fig. 7), and when the prostatic urethra has been divided at its vesical end with scissors or knife (Fig. 7*b*), this mass can be removed *en masse* on the shaft of the prostatic tractor (Fig. 7c). Tactile and visual inspection of the vesical neck is now easy, and tags or defects may be remedied by the proper procedure (Fig. 7*d*). Small

4. In ninety cases we have had but one rectal injury, caused by an inexperienced assistant holding the retractor so that its edge dug into the rectal wall. This fistula closed promptly and spontaneously. We have not one case of incontinence from injury to the external sphincter.

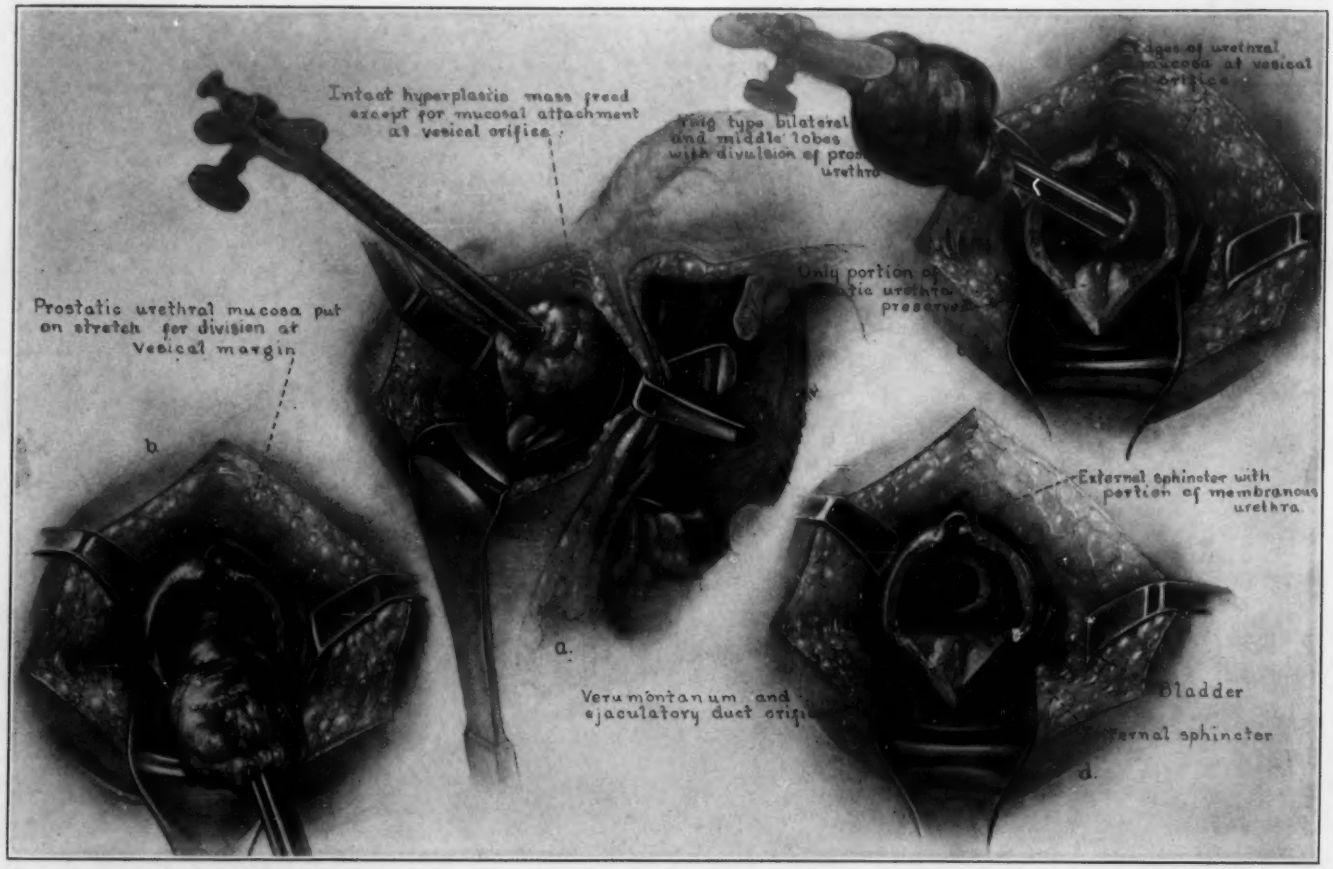
Intact hyperplastic mass freed except for mucosal attachment at vesical orifice
Prostatic urethral mucosa put on stretch for division at vesical margin
b
a.
Verumontanum and ejaculatory duct
External sphincter with portion of membranous urethra
Bladder
d.

Fig. 7.—Method of enucleation: *a*, the hyperplastic mass has been freed in its prostatic bed except for its mucosal attachment at the vesical end; *b*, the urethral mucosa put on a stretch at the vesical margin preparatory to its division with knife or scissors; *c*, ring type hyperplastic mass slid out on tractor shaft after division of the mucosa as shown in *b*. The greater portion of the prostatic urethra has been removed with it. The ragged edges are seen at the vesical margin. Internal sphincter has been surgically preserved; the structural result of perineal *en masse* enucleation is shown at *d*. Prostatic urethra divulsed; internal and external sphincters preserved; ejaculatory ducts and verumontanum preserved; healing is possible with almost complete, or complete, obliteration of the prostatic cavity and for this healing the methods of drainage and of packing for hemostasis are important.

subcervical or intravesical lobes which failed to come away with the main mass can now be accurately and cleanly removed.

The enucleation having been completed, a catheter on a stilet is passed through the urethra by an assistant, and its end is inserted into the bladder alongside the tractor, which is then removed (Fig. 8). The catheter is thus placed for reestablishment of the urethra as is done in external urethrotomy. A second catheter is placed through the perineum into the bladder for the purpose of through-and-through irrigation. The prostatic cavity is lightly packed about the two catheters passing through it, with one strip of gauze (Fig. 8*b*), which is brought out with the perineal catheter at one side. The triangular flap of the posterior lobe, carrying the ejaculatory ducts and verumontanum, is replaced in position by a mattress suture of chromic gut on the side opposite the gauze and catheter (Fig. 8*c*). An infraprostatic gauze pack is placed for the purpose of cavity obliteration and hemostasis (Fig. 8*d*). Closure now follows the usual technic (Fig. 9.) The gauze and perineal catheter are removed after twenty-four hours. It is our custom to remove the prostatic pack first, and, if no bleeding follows, in one-half hour the infraprostatic pack with the perineal catheter is removed. If there is bleeding, it is readily controlled by pressure with hemostat against the infraprostatic pack, and repacking is not required. The urethral catheter is often left in from four to twelve days, and suction applied to it by a Connell apparatus or Davis bottle. In ten of the twenty-five cases the perineum remained dry after the second day and healed, to all practical purposes, by first intention.

COMMENT

This type of inverted V incision of the posterior lobe is not new as it was used by Young in his earliest cases but has been replaced by two lateral incisions because it was thought that better anatomic preservation was thus possible. Any systematic effort for clean, complete removal of the enlarged prostatic mass in one piece through the perineum, however, has not been previously made. With medium-sized enlargements of glandular type, removal through lateral incisions is satisfactory, and remarkable preservation of the mucosal layer of the prostatic urethra, leaving the verumontanum and ejaculatory ducts intact, is possible. But in enucleating large glands, tears and extension of the lateral incisions to the membranous urethra almost invariably result, and this leaves a very narrow strip of tissue as the ejaculatory bridge. The ejaculatory ducts and verumontanum can be satisfactorily preserved with the V type of posterior lobe incision irrespective of the size and pathology of the hyperplasia. A good view of the vermontanum can always be had, and the necessary amount of mucosa for its preservation can be trimmed off with knife or scissors from the supra-

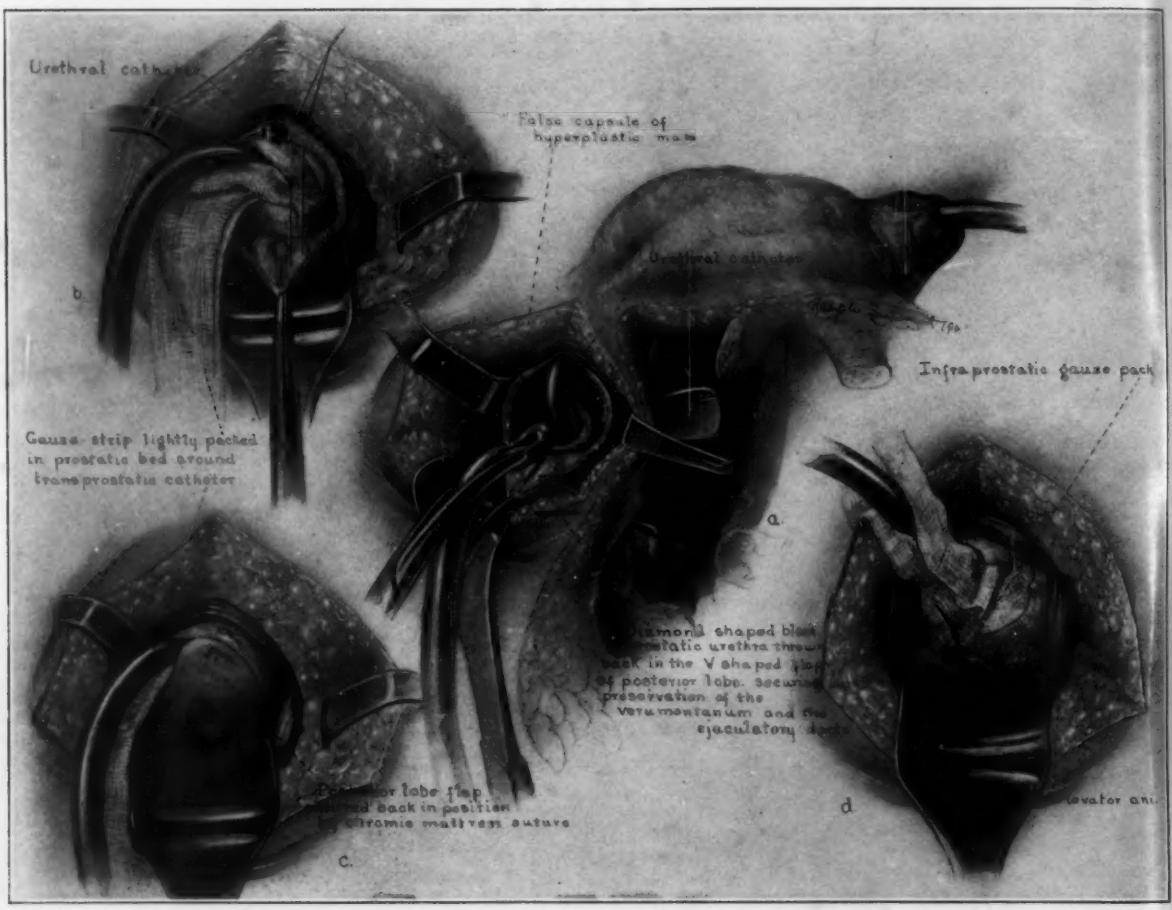

Fig. 8. — Method of catheter drainage and packing; *a*, urethral retention catheter is placed for vesical drainage and reestablishment of prostatic urethra. Perineal catheter is placed for drainage only; *b*, light prostatic gauze pack in the prostatic bed for hemostasis; removed in twenty-four hours; *c*, closure of the prostatic flap by a mattress suture on the left with gauze and perineal catheter issuing through right sided slit; *d*, placement of a large or small infraprostatic gauze pack which is removed one-half hour after the prostatic gauze is removed provided there is no hemorrhage.

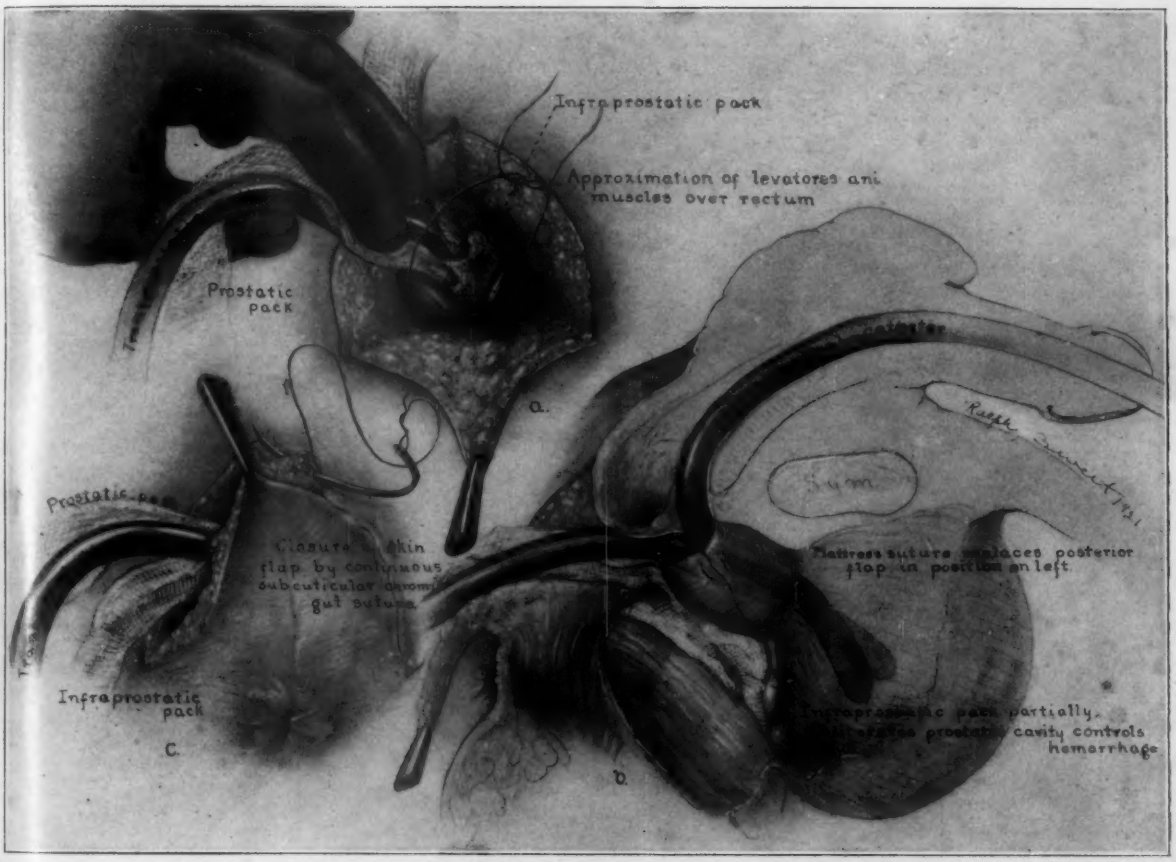
Infraprostatic pack
Approximation of levatores ani muscles over rectum
Prostatic pack
a.
Prostatic pack
Closure of skin flap by continuous subcuticular chromic gut suture
Infraprostatic pack
c.
Mattress suture replaces posterior flap in position on left
Infraprostatic pack partially obliterates prostatic cavity controls hemorrhage
b.

Fig. 9.—Method of drainage and packing: *a*, protection of the rectum by approximation of levatores ani between it and infraprostatic gauze pack; *b*, urethral catheter establishes the prostatic urethra and the infraprostatic pack obliterates the prostatic cavity; *c*, method of closing skin with subcuticular chromic gut. The catheter and gauze strips have an exit at one side. The former anchored with silk suture, the latter marked so that the prostatic pack can be first removed.

Fig. 10.—Ring type of bilateral and middle lobe enlargement removed in recent case through the perineum. Small fibromyo-adenoma enucleated *en masse* with complete preservation of colliculus and internal sphincter, but with removal of the supramontane portion of the prostatic urethra.

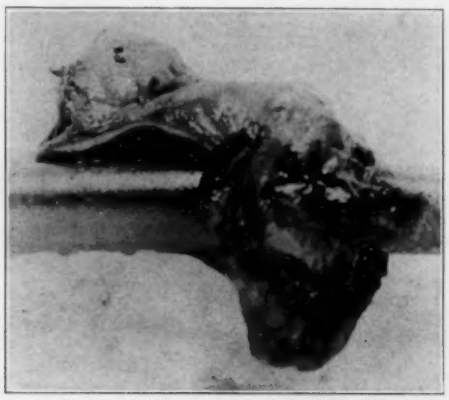

Fig. 11.—Side view, showing long mass of hyperplasia extending intravesically posteriorly to form the middle lobe.

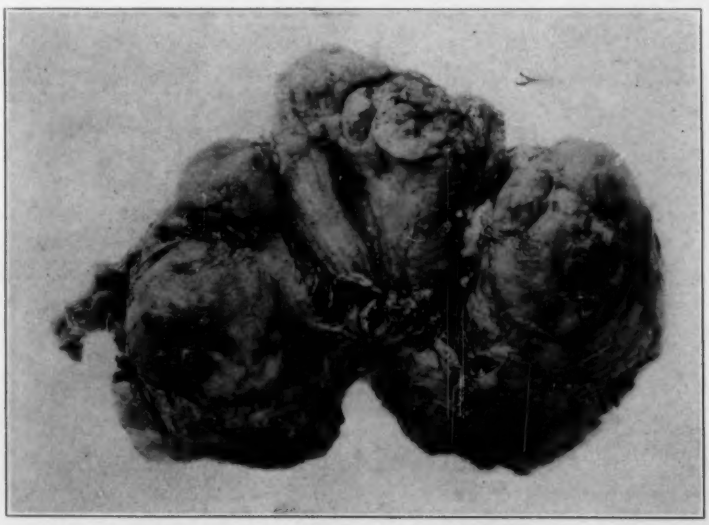

Fig. 12.—Large bilateral and middle lobe removed by way of the perineum in Case 13 (reported in accompanying table). Glandular type hyperplasia with clean-cut separation, anteriorly, of lateral lobes which fuse with middle lobe posteriorly.

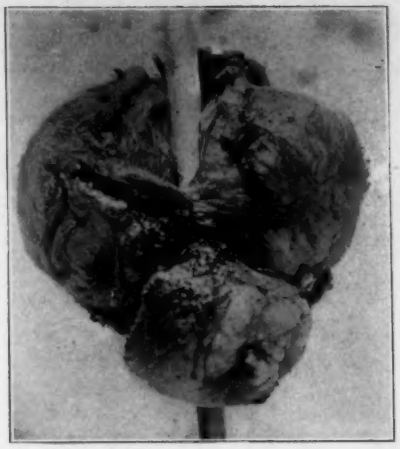

Fig. 13.—Large glandular ring type hyperplasia, enucleated intact by way of the perineum before the importance of incision of the supramontane portion of the prostatic urethra was appreciated. The colliculus and terminal portions of the ejaculatory ducts have been removed with the prostatic urethra.

Fig. 14.—Posterior urethra spread apart to show verumontanum; same case as shown in Figure 13.

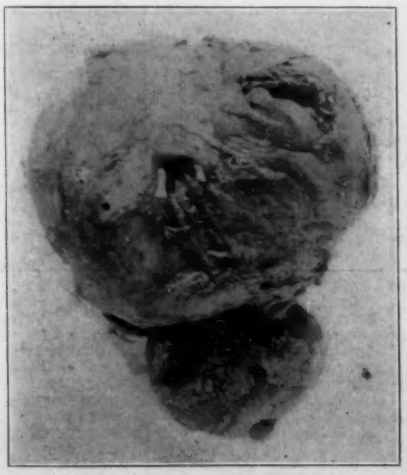

Fig. 15.—Posterior view showing distal ends of torn ejaculatory ducts; same case as shown in Figure 13.

Serial number	1	2	3	4	5	6
I Urination:						
Give the average number of voidings during the day	6	4 to 6	Every two to four hours	3 times	4-5 times	About every 2 hours
Do you get up at night to void?	Yes	Never more than once, sometimes none	Yes	Yes	No	Yes
Give the average number of times	2	.....	Twice	Once or twice	0	Three hrs.
Give the approximate time of night that these voidings occur	2 a. m.	2 to 4 a. m. or not at all	2:30 a. m.-6 a. m.	Towards morning	.....	.....
Do you have burning on urination?	No	No	No	No	0	No
Do you have hesitancy of urination?	No	No	Yes with little dribbling	No	0	No
Do you have difficulty of urination?	No	No	None	No	0	No
Do you have dribbling of urine?	Yes	No	Yes	No	0	No
1. Is this at end of urination?	Yes	.....	Yes, before and after	.....	.....	.....
2. Is this between urination?	Yes	.....	Occasionally on exertion	.....	.....	.....
Do you have good control of urination?	Fairly good	Yes	Except as above stated	Yes	Very good	Yes
What is the character of your urine:						
Clear?	Clear	Clear	.....	Clear	Yes	Clear
Or cloudy?	.....	.....	Slightly	.....	.....	.....
II. Regarding Your Sexual Life Since Operation:						
1. Have you sexual desire?	Yes	Yes	Yes	No, not often	Occasionally	Yes
2. Do you have erections?	No	Yes	No, my operation completely emasculated me	No	No	.....
A. Are they satisfactory?	.....	Yes	.....	.....	.....	Not very
B Or unsatisfactory?	.....	.....	.....	.....	.....	Unsatisfactory
3. Have you had sexual intercourse?	No	Yes	If penetration is essential, no	Seldom	No	Yes
A. If not, has it been attempted?	Yes	.....	Attempted twice since leaving hos. Decidedly unsatisfactory. There was seminal discharge but incomplete and unsatisfactory	Seldom	No	.....
B. Or not attempted?	.....	.....	.....	.....	.....	.....
C. Give average frequency and character	.....	Once in two weeks	.....	.....	.....	.....
4. Is ejaculation complete?	.....	Yes	.....	.....	.....	.....
5. Do you have seminal discharge?	No	Yes	As stated above	No	No	.....
III. General Condition:						
1. Do you consider yourself cured of your prostatic trouble?	Yes, with exception of the dripping	Yes	Yes, as far as ability to void urine	Yes	Yes	Yes
A. If you do not, please describe the character of present disability	.....	Loss of sexual power	.....	.....	.....	.....
2. How does your general health compare with what it was previous to operation?	Good, only I am suffering from neuritis which I had before operation and I get pretty bad at times which you can see by my writing as I can hardly hold my pen	Very good	It is somewhat improved. I have good physical endurance, can walk and drive my auto long distances, 200 miles at one sitting without fatigue	Much better	Fully as good	Good
Remarks	Catheter life for 1 year before operation. Has been seen since above answers were received and "dripping" is insignificant	Periodic catheter life for 3 years	Coudé catheter broke off in prostatic urethra after operation and fragment had to be removed on 5th day per perineum followed by pronounced urinary infection with bilateral epididymitis; had intermittent catheter life for 6 months before operation	Man of 88 had a huge prostate and left hospital wearing a urinal on account of dribbling. Final result is a complete cure. Had led catheter life for 5 months	Very large prostate in man of 76. Catheter life for 6 months	Complete retention and marked vesical distention on admission, 5 weeks preparatory treatment. Had led catheter life for 3 months

7	8	9	10	11	12	13	14	Summary
5 to 6	4	4-5	3 to 4 hrs.	4 to 5	3 to 4 times	About every 4 hours	4 times	4 to 5
Yes	No	Yes	Yes	No	Yes	Yes	Yes	No, 3 Yes, 11
Always once sometimes twice	No	2-3, get up for bowel movement void at same time	3 to 4 times	.....	6 to 7	Two	2	2
6 a. m.	.....	.....	.....	.....	.....	12 and 4	2-5 a. m.	
No	No	No	I do	No	No	No	No	No, 13 Yes, 1
No	No	No	No	No	No	No	Occasionally	No, 12 Yes, 2
No	No	No	No	No	No	No	No	No, 14
Slight in morning	Slight	No	Yes	No	No	No	No	No, 9 Yes, 5
Yes	Yes	.....	No	.....	.....	.....	.....	4
No	No	.....	Yes	.....	.....	.....	.....	3
Yes, very good	Yes	Yes	Fairly good	Yes	Yes	Yes, very good	Yes	Yes, 14
Yes	Yes	.....	.....	Yes	Yes	.....	Yes	10
.....	.....	Occasionally	Cloudy, strong odor	.....	.....	Yes	.....	4
Sometimes	Yes	.....	No	No	.....	Practically none	Yes	9
Partial	Few	.....	Very little	No	No	No	Yes	6
No	No	.....	No	.....	.....	.....	Yes	No, 4 Yes, 3
Yes	.....	.....	Yes	.....	.....	.....	.....	4
No	Yes	.....	No	No	No	No	Yes	5
No	....	.....	No	No	No	No	.....	2
.....	.....	.....	.....	Not attempted	.....	Yes	.....	6
.....	Once or twice a week	.....	.....	.....	.....	.....	Once every two weeks	
None	No	.....	.....	.....	.....	.....	Yes	2
Not attempted. No intercourse 2 yrs. before operation	No	.....	Yes	No	No	No	Yes (watery nature)	3
Yes	Yes, but in voiding the urine comes freely for a while but sometimes shuts it off before I get through and there is a dribbling for a time	Yes	Yes	Yes	Yes	Yes	Yes	14
Excellent much better than before operation	Decidedly better	Improved	Much better	Greatly improved	My health is as good as before operation, my only trouble is that my legs are very weak from thigh to knee	Much improved	Very much improved	Improved in 12 As good in 2
Preliminary cystotomy and removal of large vesical calculus. Periodic catheterization for 3 months	This man has since been cystoscoped and a small tag of mucous membrane at the vesical margin removed by fulguration with complete relief of dribbling and hesitancy. Had complete retention before operation and used catheter 3 months	This patient has senile dementia which was present before operation	Had pronounced pyuria and bilateral pyelonephritis with cystitis before operation	Catheter life for 1 year before operation	This patient has a chronic nephritis and cardiac decompensation. Under preparation for 6 weeks. Had periodical incontinence on admission	Catheter life for 4 months before operation	Intermittent catheter life for 6 months	All were advanced cases of prostatism and had been compelled to use a catheter intermittently or regularly

montane portion, so that when this is removed later, with the hyperplastic mass, tearing of the vermontanum and consequent disturbance of the ejaculatory ducts do not occur. Before we appreciated the importance of the deep incision in this supramontane portion of the prostatic urethra, in two or three instances the verumontanum with the ends of the ejaculatory ducts was torn away with the hyperplastic mass (Figs. 10 and 11). These experiences lead us to believe that the colliculus and the terminal portions of the ejaculatory ducts are often similarly removed in suprapubic *en masse* enucleation.

Attempts to preserve the membranous coat of the prostatic urethra when one follows Young's technic in patients with big prostates are usually unsuccessful. In view of the deep sulci and the distortion and elongation of the prostatic urethra in these cases, preservation seems undesirable. So much loose unattached mucous membrane may be so folded or plicated that it forms obstruction to instrumentation as well as urination and prevents good structural healing. The ability of urethral mucosa to repair is familiar to every urologist who can readily recall extensive resections of the urethra in performing external urethrotomies and the rapidity with which these gaps fill in. Complete divulsion of the prostatic urethra is universally practiced in suprapubic enucleation, and it ought not to be more mutilating when done perineally under direct control of the eye. However, is such complete divulsion of the prostatic urethra responsible for the large cavity formation so much more frequently encountered after suprapubic prostatectomy? This is doubtful. Cavity formation is more readily accounted for by the difference in methods of packing for the control of hemorrhage and the anatomic difference of enucleation. Suprapubically, the whole enlargement must be shelled out through the internal vesical sphincter, and often the vesical mucosa is stripped back from the bladder wall. Perineally, only the intravesical portion of the gland is drawn back through the sphincter, which is undisturbed surgically. The suprapubic operator packs the cavity full from above, elongating and exaggerating the prostatic space and often adding to the separation of the vesical margin from the prostatic cavity. The perineal operator packs lightly and partially from below and relies largely on infraprostatic compression for hemostasis, which tends to foreshorten the prostatic space and obliterate the cavity. Repacking suprapubically only exaggerates and prolongs the existence of the cavity. Repacking through the perineum is almost invariably an infraprostatic compression with further obliteration of the cavity. In none of our twenty-five cases of *en masse* enucleation by prostatic urethral divulsion through the perineum has cavity formation ever occurred.

The great advantage of this modification is that it secures a more complete removal of the hyperplasia. Ring type enlargements are quite frequent, and even if they are not typical, there

is some fusion of tissue between the separate lobes (Fig. 13), and it is an easy and no uncommon matter to leave behind small or large spheroids at the region of separation of these lobes when they are removed separately or in portions. The smaller fibrous enlargements, particularly, tend to be ring type, and resection of such enlargements is extremely difficult and incomplete when performed by segments, but much simpler and more certain when removed *en bloc* (Fig. 12). The absence of a pseudocapsule makes such removal very unsatisfactory suprapubically, but is an insignificant factor perineally because of the good exposure obtained by laying the posterior urethra wide open, with the inverted V type incision. There is no comparison between the accuracy and ease of *en masse* enucleation by the two methods. Perineally, with blunt dissector, scissors or knife, one can perform an accurate surgical resection under direct control of the eye.

The cases that have been treated by this modification have been reported elsewhere.[5] The importance and perfection of certain details of operative technic and of methods of drainage and packing were not fully realized in the first ten or twelve cases. In the accompanying table, however, the actual answers to a questionnaire are given by these earlier patients in whom one year or more has elapsed since operation and in all of whom radical *en masse* enucleation was performed. All fourteen state that they are cured of prostatism; two have complete preservation of sexual function, and in five others, it is diminished and partial, untried in seven.[6]

The immediate results of the more recent twelve cases, in which the perfected details of the procedure were applied, have all been completely satisfactory. The average stay in the hospital after operation has been twenty-three days, and the average time for closure of the perineal fistula has been five days. Seven cases have closed to all practical purposes by first intention, there being no urinary leakage after a few hours from the time of removal of the perineal drain. In two cases, however, owing to urethral and urinary infection, the urethral catheter had to be removed early and closure was delayed, and by granulation being complete on the fourteenth and nineteenth day, respectively. One patient whose perineum healed by first intention had quite a profuse secondary hemorrhage on the ninth day, which acted and was treated similarly to the hemorrhage following a punch operation. The convalescence was uneventful in six cases; two patients

5. Cases 30 to 50, inclusive, and Case 11 of Group A; Cases 38, 39 and 40 of Group B, "Suprapubic Versus Perineal Prostatectomy" read at the Montreal meeting of the American Urological Association, June 2, 1921.

6. Before operation, sexual life was stated as normal in only four of the fourteen cases; the other ten having markedly diminished, lost or unknown, because untried, sexual ability.

had troublesome cardiac insufficiency, one renal insufficiency, two a mild phlebitis and six rather marked pyuria, with evidence of urinary infection. Only two patients had epididymitis after operation. The general and functional result indicates a complete cure in all these recent cases. Five patients have stated that their sexual life was normal before operation. It was lost or absent in two, diminished in three and not stated in two. It is already known that in three, normal sexual life has been resumed.

CONCLUSIONS

Failure of complete restoration of function after prostatectomy is commonly associated with structural defects. Suprapubically, the common defect is the persistence of a large prostatic cavity. Perineally, the defect is commonly due to nodules or lobular remnants from incomplete or irregular enucleation. It is possible to perform a one-piece perineal prostatectomy, remove the hyperplasia with the associated prostatic urethra completely and cleanly, and at the same time surgically preserve both vesical sphincters and the colliculus and ejaculatory ducts. The structural result in such a radical procedure is surprisingly good, and less apt to be defective than when done suprapubically. A large prostatic cavity has never resulted, and the prostatic urethra becomes quickly reestablished. The functional results have been entirely satisfactory in twenty-five cases.

RELAXED PELVIC FLOOR: END-RESULT IN SIXTY CASES*

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INTRODUCTION

The purpose in undertaking this study was to determine what operative procedures, in cases of relaxed pelvic floor, have given the best results in this clinic. Between the years 1913 and 1920, 130 patients were operated on for some form of relaxed pelvic floor. The nomenclature used was varied: cystocele, rectocele, prolapse, procidentia and relaxed pelvic floor. Of these, sixty patients reported in person for subsequent examination, twenty-nine reported by letter and forty-one could not be located. The cases of the sixty patients that were examined are reported in full, and certain deductions are drawn from the results in these patients. The results in the twenty-nine cases as given by letter or by talking with patients who refused examination are reported separately; but no deductions are made, as such observations appear unreliable. It is our opinion that only careful physical examination, after a suitable period of time has elapsed, should be used to judge the results achieved. In fact, several women returned for observation stating that they had been fully relieved, examination of whom showed that the cervix was protruding several inches from the labia. No attempt was made to select cases, and we believe that the patients returning for examination illustrate well the various types of this disability.

The extensive literature which pertains to this subject is evidence that the present methods of treatment are not entirely satisfactory. Articles appear frequently describing an improved operation or the variation of some older technic. The discussion of the principles and mechanism involved in prolapse is equally prolific. At the same time, it can be said that end-result studies are much less common, although it seems that careful studies of such results might eliminate at least a portion of the methods of cure variously advocated. The recent contribution of J. G. Clark[1] is an excellent example of the value of such a study.

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1. Clark, J. G.: Surg. Clinics N. America **1**:77-100 (Feb.) 1921.

Relaxation of the pelvic floor appears to be the mechanism by which all these conditions are produced. The further titles of cystocele, rectocele, prolapse and procidentia merely represent the extent of the lesion. The result of relaxation of the pelvic floor is a hernia so lucidly described by Moschcowitz [2] in his study of prolapse of the rectum. This conception of his, admirably described and illustrated with charts, he in turn derived from the anatomic study of Waldeyer [3] and the clinical work of Ludloff,[4] Zuckerkandl,[5] and others. He discovered later that the operation he perfected had been previously performed by Duval and Quénu.[6] Jones,[7] Frank [8] and Clark [1] have applied this theory of herniation to prolapse of the vagina and uterus, giving all credit to Moschcowitz for the clinical application of the idea. The condition thus simulates hernia elsewhere.

The etiologic factors may be and are many, just as with other types of hernia; but congenital defects and trauma stand out as the principal causes, and one or both are usually present. Congenital conditions undoubtedly constitute a considerable factor, and in the literature cases of nulliparas with complete procidentia are reported. Jones [7] states that the acquired form of deep culdesac is more common than the congenital type. This congenital type he thinks is due to absence or weakness of the transversalis fascia. Trauma is probably of greater importance, although there are patients even in this small group who have had between six and ten children and who present marked laceration of the perineum without any real herniation. The factor of increased intra-abdominal pressure, whether due to tumor, ascites or straining at stool with constipation seems to be of only contributary importance. According to the type and extent of the herniation, the bladder, uterus, rectum or bowel may rest within the prolapsed part.

In order to obviate descriptions of the types of operation performed, we are submitting here a short description of the various procedures used in this clinic. The fact that the staff of this hospital is small permits of such a standardization.[9] However, all experienced in gynecologic surgery will appreciate that with each case there may be some variation from standard types of operation. The number of such

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2. Moschcowitz, A. V.: Surg., Gynec. & Obst. **15**:7-21, 1912.
3. Waldeyer, W.: Das Becken, Bonn **23**:148, 1899.
4. Ludloff, K.: Arch. f. klin. Chir. **59**:447-457, 1899; Ibid. **60**:717-811, 1899.
5. Zuckerkandl, O.: Deutsch. Ztschr. f. Chir. **31**:590-608, 1891.
6. Duval and Quénu: Cited by Moschcowitz, Footnote 2.
7. Jones, D. F.: Boston M. & S. J. **175**:623-627 (Nov. 2) 1916.
8. Frank, R. T.: Surg., Gynec. & Obst. **24**:42-60 (Jan.) 1917.
9. Operations in this series were performed by the visiting surgeons, Drs. Cheever and Homans or by the successive resident surgeons, Drs. Goetsch, Jacobson and Cutler.

procedures used in such a small clinic as this is the best evidence of the necessity for such variations. There are certain principles, however, which are constantly followed.

The operations for cystocele and rectocele are performed after accepted procedures and resemble the operations so well described by Frank.[8] In the cystocele operation, after dissecting back the mucosal flaps, the bladder is freed from the cervix, pushed up and held in position by approximation of the paravesical fascia, including the pubocervical ligaments, after which the excess of mucosa is excised and the mucosa reapproximated. In the rectocele operation, after dissecting back the mucosal flaps, the levator muscles are exposed and approximated by interrupted mattress sutures, placed, to begin with, as high inward on the rectum as possible. The excision of excess mucosa and reapproximation of mucosa and skin over the perineum varies with each case. These simple plastic repair procedures have on the whole proved satisfactory.

The abdominal operations have varied more widely. The simplest types are ventral suspension and ventral fixation of the uterus. The procedures most commonly followed are the Gilliam and Olshausen methods, well described by Graves[10] in his textbook of gynecology. In performing the Gilliam suspension, our method of securing the round ligament is to pass a curved clamp from above downward through the internal inguinal ring, grasp the ligament beneath its peritoneal coat and drag it up on the anterior surface of the recti muscles, where it is sutured to the anterior rectus sheath and sometimes to the other ligament. This is essentially the Mayo[11] modification. Ventral fixation in this clinic is performed by passing either silk or chromic catgut sutures into the fundus and then above the anterior rectus sheath, tying the knot, sometimes intraperitoneally and sometimes on the rectus sheath.

Supravaginal hysterectomy, with suture of the round ligaments to the cervical stump for support or fixation of the stump to the anterior abdominal wall, is the most frequent procedure for prolapse of the uterus. Graves finds this a most satisfactory operation, with which opinion our statistics concur. In some cases, one ovary has been tucked into the broad ligament.

Bisection of the uterus with removal of the endometrium and suture of the two halves of the uterus to the rectus sheath on each side has been attempted in several of the most pronounced cases of procidentia.

The Mayo operation of vaginopexy is also recorded a few times, performed both by the vaginal and abdominal routes.

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10. Graves, W. P.: Gynecology, Philadelphia, W. B. Saunders Company, 1917.

11. Mayo, W. J.: Surgical Treatment of Prolapse of the Uterus and Walls of the Vagina, J. A. M. A. **59**:1421-1424 (Oct. 19) 1912.

An operation in which we are greatly interested and which we have called here the Moschcowitz procedure is an adaptation of Moschcowitz's operation for prolapse of the rectum, first used for this purpose, we believe, by Dr. D. F. Jones,[7] of Boston. Clark [1] also reports much success with, and interest in, this operation. In performing this operation, the uterus is drawn up and the pouch of Douglas obliterated by spiral stitches commencing on the lateral and posterior walls of the pelvis and running over to the cervix, final sutures being taken to approximate the uterosacral ligaments. When these sutures are completed, the cervix is held at a high level, and enough space is left posteriorly, at the entrance to the pelvis, for passage of the rectosigmoid. The posterior culdesac should thus be obliterated. The fundus is then held upward and forward by some form of ventral fixation or suspension. If future pregnancies are desired, the suspension may be of the Gilliam type. We have had such patients return after childbirth (Case 10, Chart 2). This principle of posterior fixation of the cervix and obliteration of the culdesac may, indeed, be applied following supravaginal hysterectomy. On the whole the logic of, and results achieved by, this operation are extremely creditable.

The interposition principle has not been used in operations in this clinic.

PRESENTATION OF MATERIAL

Some classification seemed necessary in order to present the results more clearly. A classification has been selected based on the age and child-bearing possibilities of the patient rather than on the type and extent of the lesion. Of necessity the treatment must vary both according to the desire for more children and because of the severity of the lesion, for one cannot invariably consider such lesions from the ideal point of view of what method will give a more nearly permanent cure.

The following arbitrary grouping appeared most convenient: Group 1, women in the child-bearing age; Group 2, women past the child-bearing age. The dividing line cannot correspond to the biologic limit, since it seems reasonable to include in Group 2 women near the menopause who do not desire more children and those with serious organic diseases, such as cardiac or renal disease, in whom future pregnancies would be a serious menace to life. Each group is further subdivided into Class A, vaginal prolapse (cystocele and rectocele), in which the uterus is in its normal relation; Class B, vaginal and uterine prolapse in which there is some degree of uterine descensus and Class C, procidentia, in which there is uterine prolapse with the uterus outside. There is nothing new in this method of grouping and classification, and there is little to commend it, except the fact that the question of future child-bearing warrants a definite limit and that the extent of

the lesion gives a fairly definite syndrome, the variations of which may demand varying types of operation. It is understood, however, that in the final consideration as to the wisest operative procedure each case demands its own analysis. Nevertheless, this scheme provides a convenient skeleton for purposes of discussion.

Of the total 130 patients operated on and used as the basis of this study, forty-seven are to be considered as falling in Group 1 and eighty-three in Group 2. Their ages varied from 23 to 80 years. All but four were married, and the number of pregnancies in each case varied from none to thirteen. The number of pregnancies, however, has not appeared to exert much influence on the type or degree of lesion. The four unmarried women suffered from marked prolapse and procidentia, and prolapse was equally common among those with few or many children. Equally striking is the fact that age seems to be of no significance. There are among these cases older women with ten, twelve and thirteen children who showed simple cystocele or rectocele with

TABLE 1.—NUMERICAL DIVISIONS OF CASES

Class	Group 1	Group 2
A. Vaginal prolapse only	6	7
B. Vaginal and uterine prolapse	12	26
C. Procidentia	0	9
	18	42

some laceration of the perineum, while women in the second and third decades with one or two children showed complete procidentia. These facts seem to indicate that there is frequently some congenital predisposition to this lesion and that child-bearing and age are in certain cases only secondary factors similar to increase in intra-abdominal pressure and differing only in degree.

A. PATIENTS RETURNING FOR EXAMINATION

The chief interest is centered on the sixty patients who returned for examination. This includes only patients seen after an interval of at least six months following operation. The average interval was thirty-five months, the interval varying from six to ninety-one months. In only six cases was the interval from operation to final observation under one year, namely, six, eight, nine, ten, eleven and ten months. Of the sixty patients, eighteen are in Group 1. All of these eighteen patients were under 40 years of age and desired more children or were so young that it was thought that sterilization or further abdominal operation was unjustifiable. This leaves forty-two patients in Group 2.

The numerical divisions of cases into groups and classes is given in Table 1.

The excess of patients in Group 2 over Group 1 will doubtless be taken by some to contradict any statements regarding the small part age and trauma play. We believe, however, there is a better explanation in the following facts: First, relaxation of the pelvic floor, like inguinal hernia, is a progressive lesion, and many patients seek relief only when time has proved the futility of hoping for improvement and has shown the increase in their disability. Second, women with milder degrees of this trouble, when still in the child-bearing age, are often advised to wait until this period of life is over before entering the hospital. This means that many women with milder degrees of herniation who would enter the hospital in Group 1 are turned away temporarily and on return subsequently will fall within Group 2.

All but one patient in Group 2 were married. The average age in Group 1 was 33 and in Group 2, 48. The average number of children in Group 1 was 2.5, in Group 2 was 4.6.

The following tables summarize the material in full by groups and classes. Table 2 deals with the cases of vaginal prolapse in Group 1. These patients all suffered from mild degrees of vaginal prolapse, two patients with rectocele alone and four patients with rectocele and cystocele. Two patients had a badly torn perineum. All these women were married and had had children at the time of operation, and none are reported as having had subsequent pregnancies. With one exception, they were in good general physical condition. Symptoms had been present for from nine months to eight years, and in the great majority of cases they dated back either to the last childbirth or the one preceding the last. The chief complaint varied from simple backache, which was present in most cases, to moderate bladder or bowel incontinence and frequently to an actual protrusion from the vagina.

All patients but one were subjected to relatively simple operative procedure. All had local plastic operations, and only one of the six had an abdominal operation in addition. The results on the whole are fair, and no patient showed a complete recurrence though the interval from operation to final observations averaged forty-six months, more than three and one-half years.

The most radical operation, Case 1, and one of local repair, Case 3, gave excellent results. The radical operation, hysterectomy, was performed because of complicating fibroids rather than on account of the greater degree of the local lesion. If it is true that herniation with a congenital weakness is behind many cases, one would expect that cases of this class in Group 1, with simple vaginal prolapse for which vaginal plastic operations were performed, would progress and in time recur, even as a simple inguinal hernia, with muscle and fascia repair but

TABLE 2.—Group 1, Class A, Vaginal Prolapse, Six Cases

Case; Surgical No.	Married or Single; Age; No. of Children	Preoperative Condition	Previous Operation	Local Condition	Complicating Condition	Chief Complaint; Duration	Operative Procedure	Months to Final Observation	Result at Final Observation
1 11025	Married 35 4	Good	None	Small cystocele; large rectocele	Fibromyomas of uterus	Backache; 2 years	Anterior and posterior colporrhaphy; perineorrhaphy; supravaginal hysterectomy*	20	Firm perineum; some right sided pain; good result
2 1998	Married 38 1	Good	None	Large rectocele; small cystocele	None	"Falling of womb and backache"; 6 years	Anterior and posterior colporrhaphy	69	Slight cystocele; functional incontinence; cervix well supported
3 2296	Married 26 3	Good	None	Large rectocele; small cystocele	None	Weakness, 4 years	Dilatation and curettage; anterior and posterior colporrhaphy	16	Good perineal body; small movable uterus; well supported
4 5432	Married '38 3	Good	None	Small rectocele	Cervical polyp	Lump in vagina; 4 years	Posterior colporrhaphy; amputation of cervical polyp	51	Firm perineum; small outlet; uterus 1 degree retroverted
5 573	Married 38 2	Good	None	Small rectocele; complete tear of perineum	Dysentery	Bowel incontinence, relaxed vagina; 4 years	Posterior colporrhaphy, repair of complete tear	81	Some descensus; slight retrocele; uterus well supported
6 4962	Married 33 13	Poorly nourished	None	Large rectocele; small cystocele; lacerated perineum	Fibroid of uterus (?)	Menorrhagia, pain right side; 9 months	Dilatation and curettage; anterior and posterior colporrhaphy	40	Perineum fair; menorrhagia; fibroid uterus diagnosed; operation advised

* Unless otherwise stated "supravaginal hysterectomy" implies "with ventral suspension of cervical stump by round and broad ligaments."

TABLE 3.—GROUP 1, CLASS B, VAGINAL AND UTERINE PROLAPSE, TWELVE CASES

Case; Surgical No.	Married or Single; Age; No. of Children	Preoperative Condition	Previous Operation	Local Condition	Complicating Condition	Chief Complaint; Duration	Operative Procedure	Months to Final Observation	Result at Final Observation
1 8243	Married 36 5	Good	None	Large cystocele; small rectocele; lacerated cervix; retroversion	None	Pain in back, falling of womb; 12 years	Anterior and posterior colporrhaphy, supravaginal hysterectomy, bilateral salpingectomy, incidental appendicectomy	21	Cervix high, good perineum, good result
2 8680	Married 39 2	Obese	None	Large rectocele; small cystocele; retroversion	None	Falling of womb; 6 years	Amputation of cervix, posterior colporrhaphy, ventral suspension by Gilliam method, incidental appendicectomy	24	Feels fine; no rectocele or cystocele, cervix high; good result
3 6990	Married 37 13	Good	None	Large cystocele; small rectocele; retroversion	None	Pain in lower abdomen; 3 years	Anterior colporrhaphy; ventral fixation of uterus	36	No cystocele or rectocele; uterus well suspended; dysmenorrhea; good result
4 941	Married 31 2	Poorly nourished	Dilatation and curettage	Small rectocele; moderate descensus	None	Womb trouble, pain lower abdomen; 7 years	Dilatation and curettage, posterior colporrhaphy, ventral suspension (Gilliam)	17	Slight bulging of vagina; cervix well suspended; complains of piles; good result
5 130	Married 33 2	Good	None	Small rectocele; retroversion	None	Falling of womb; 3½ years	Anterior and posterior colporrhaphy, ventral suspension (Gilliam)	91	Perineum strong; no complaints; good result
6 3173	Married 39 2	Good	Dilatation and curettage 12 years ago	Large cystocele; small rectocele; descensus	None	Falling of womb, backache; 6 years	Amputation of cervix, anterior and posterior colporrhaphy	26	Complains of recurrence, 3 mos.; much frequency of urination and dysuria; feels miserable; moderate descensus and recurrence of rectocele and cystocele

7 10420	Married 27 2	Good	None	Moderate cystocele and rectocele with descensus	None	Bulging from vagina, descensus, functional incontinence of urine; 9 years	Dilatation and curettage, anterior and posterior, colporrhaphy, ventral suspension (Gilliam)	8	Cervix protrudes from vagina; return of symptoms
8 6744	Married 32 2	Good	None	Small cystocele and rectocele with retroversion	None	Lower abdominal pain; 2 months	Amputation of cervix, posterior colporrhaphy, ventral suspension (Gilliam)	11	Dysuria, lumbar backache; uterus in good position with slight tenderness in vaults
9 6215	Married 37 3	Good	None	Lacerated perineum and cervix, retroversion	None	Backache; 8 years	Trachelorrhaphy, posterior colporrhaphy, ventral fixation, incidental appendicectomy	41	Slight cystocele and rectocele, good perineal body, cervix well supported, only fair result
10 8007	Married 22 2	Good	None	Lacerated cervix and perineum, prolapse	None	Dragging sensation in pelvis, backache, 1½ years	Trachelorrhaphy, posterior colporrhaphy, Mosheowitz with Gilliam suspension	40	Child born one year following operation, with backache since; examination shows weak perineum, no cystocele or rectocele, cervix low and anterior, fundus in normal position
11 10048	Married 32 2	Good	None	Cystocele with prolapse	Cholelithiasis	Debility, backache, indigestion, abdominal pain; 3 years	Dilatation and curettage, anterior colporrhaphy, ventral suspension (Gilliam), appendicectomy, cholecystectomy	22	Uterus in good anterior position, normal adnexa, no cystocele, return of indigestion for six months
12 8273	Married 37 2	Good	None	Prolapse of uterus	None	Debility, menorrhagia, leukorrhea, backache; 15 years	Dilatation and curettage, ventral suspension (Gilliam), bilateral salpingectomy	23	Uterus in good anterior position, slight leukorrhea

without operation on the peritoneal sac, would recur. The evidence in this small group is that local operation in early cases of vaginal prolapse effects permanent relief. The explanation may lie in the fact that if the lesion is taken early enough, and further trauma, as childbirth, does not occur, the further progression of the incipient lesion is prohibited. Another explanation is the accepted theory that trauma produces a local weakness which may result in simple local prolapse in which the tear is insufficient to initiate herniation of the pouch of Douglas. In this class of cases, the latter explanation is probably the more justifiable.

Table 3 shows the cases of vaginal and uterine prolapse in Group 1. Into this class fall patients in whom there is some degree of descensus of the uterus in conjunction with vaginal prolapse. Uterine prolapse without some degree of vaginal prolapse is uncommon. All patients were married and had children at the time of operation. Only one patient, Case 10, has reported a subsequent pregnancy. The chief complaint was either "falling of the womb" directly, or such symptoms as bulging from the vagina, and backache. The condition had been present symptomatically from two months to twelve years. At the time of operation, all patients were considered good surgical risks.

All patients but one (Case 12) were submitted to some type of local vaginal repair, associated in three cases with amputation of the cervix and in one case with trachelorrhaphy. In addition, all but one had laparotomy, the abdominal procedure being: ventral suspension by modified Gilliam operation, 8 cases; ventral fixation with silk, 1 case; Olshausen suspension, 1 case; supravaginal hysterectomy with round ligament support to the cervix, 1 case, and Moschcowitz procedure, 1 case. Case 6, in which no abdominal procedure was carried out, recurred completely after three months. Of the eleven other patients, all but one, Case 7, have done well and showed a good anatomic result at the time of the last examination. The explanation for the failure in Case 7 probably lies in some mechanical failure at, or shortly after, operation. It should be remarked that at operation in this case a large retroverted uterus was found. It is possible in a Gilliam suspension when the round ligaments are very lax that they should stretch farther and allow descensus to recur, though temporarily holding the uterus in good anterior position. The excellent results of the Gilliam operation in general would seem to show its efficacy. It may be said, however, that in this case there was more descensus than in the others, and the cervix actually came to the labia. In such cases, in degree almost procidentia, with great slackening of all supports and herniation of the pouch of Douglas, a more radical operation should be performed than any type of round ligament suspension which leaves the pelvic floor

still unprotected from the thrust from above. In Case 10 the Moschcowitz procedure was carried out; the cervix was held posteriorly; the culdesac obliterated and the fundus suspended by a Gilliam operation. Within five months, this patient was pregnant. There was a normal delivery. The final examination seemed to indicate that the posterior fixation of the cervix had broken away. The symptomatic and anatomic result, however, was good.

A study of this class demonstrates that with descensus a combined operation is necessary, and that although simple suspension may suffice, if the degree of relaxation is considerable, some radical attempt at obliteration of, or support to, the posterior culdesac is necessary. The failure in Case 6 should have been foreseen, while the failure in Case 7 suggests the need of a more careful study of the actual relaxation and herniation at the time of operation and a better adaptation of procedure to conditions found within the pelvis. The youth of this patient, 27, probably was the reason why nothing more radical was done. Furthermore, she desired more children.

There were no cases of procidentia in Group 1.

Group 1 as a whole shows satisfactory results, two cases in Class B, Cases 6 and 7 being the only failures in the eighteen cases, 89 per cent. successes. Case 7 should not be judged too severely, however, since in a patient of 27 no surgeon would undertake an operation entailing sterility unless the condition absolutely demanded it. Moreover, the Gilliam operation plus vaginal plastics is usually sufficient. The general deductions to be drawn are that before the menopause in women desiring more children, vaginal prolapse can usually be permanently relieved by plastic operations on the vagina, and that when the vaginal prolapse is accompanied by moderate uterine descensus, the condition can be relieved by vaginal plastic operations plus some simple abdominal procedure, preferably the Gilliam suspension or the Moschcowitz procedure.

As shown in Table 4, Class A, Group 2, is relatively small. All seven patients show simple vaginal prolapse; all had children. The chief complaint consisted of a bulging from the vagina or frequency of micturition. These symptoms had existed for from four months to twenty years, with an average of more than twelve years. No patients had had previous pelvic operations.

In all cases simple vaginal plastic operations were performed, combined with amputation of the cervix in one case only. The average duration of time from operation to final examination was thirty-five and one-half months, almost three years. The anatomic results at this time were good in all except one instance, Case 7. With this exception there was no real recurrence in any case, though in two cases a small

TABLE 4.—Group 2, Class A, Vaginal Prolapse, Seven Cases

Case; Surgical No.	Married or Single; Age; No. of Children	Preoperative Condition	Previous Operation	Local Condition	Complicating Condition	Chief Complaint; Duration	Operative Procedure	Months to Final Observation	Result at Final Observation
1 11125	Widow 45 5	Good	None	Large cystocele and rectocele	Hemorrhoids	Bulging from vagina; 8 years	Anterior and posterior colporrhaphy, perineorrhaphy; clamp and cautery for hemorrhoids	19	Firm perineum, no cystocele or rectocele, good plastic result; general nervousness, rapid pulse, palpable thyroid, metabolism +31, hyperthyroidism
2 11495	Married 54 3	Obese	None	Large cystocele; small rectocele	None	Bulging from vagina; 20 years	Anterior and posterior colporrhaphy	16	Excellent perineum; no cystocele or rectocele; no descensus; good result
3 5759	Widow 53 13	Good	None	Moderate cystocele and rectocele	None	Pain in the right lower quadrant; 20 years	Dilatation and curettage, posterior colporrhaphy	31	No cystocele or rectocele; occasional sharp pain in left lower quadrant; no descensus; good result
4 4798	Married 47 7	Good	Dilatation and curettage	Large cystocele	Inguinal hernia	Lump in vagina; 4 months	Anterior colporrhaphy	50	Small cystocele, no rectocele, descensus, backache
5 6797	Married 41 4	Poor	None	Moderate cystocele and rectocele, lacerated perineum	Chronic nephritis, Hypertension	Torn perineum; 5 years	Posterior colporrhaphy, repair of complete tear	37	Incontinence and dysuria; obese; diastasis recti; no cystocele or rectocele; no prolapse; good result for plastics
6 5297	Married 40 4	Good	None	Lacerated and hypertrophied cervix, slight rectocele	None	Leukorrhea	Dilatation and curettage, amputation of cervix, posterior colporrhaphy	51	Obese; backache and frequency; small cystocele; uterus well supported; fair result
7 10360	Married 40 4	Obese	None	Large cystocele, small rectocele, lacerated cervix	None	Frequent micturition; 18 years	Anterior and posterior colporrhaphy	9	Recurrence; reoperated on 10 mos. later here; anterior and posterior colporrhaphy, perineorrhaphy, posterior fixation of cervix, anterior fixation of cervix, (prolapse)

cystocele was present with some frequency of urination. One patient (Case 7) suffered a recurrence with descensus and was reoperated on ten months later in this hospital, the vaginal plastics being done again and a Moschcowitz operation performed with posterior fixation of the cervix and anterior fixation of the fundus. This case showed no recurrence eight months later.

The long duration of symptoms in this group is further evidence of why Group 1 patients are relatively few and the disproportion between Classes A and B can be taken as evidence of the progressive nature of the lesion. That some patients, however, have simple vaginal prolapse for a period of twenty years, as shown in two cases in the present group, is probably best explained by the fact that in these cases, so few as to be exceptional, the lesion is chiefly traumatic, with little congenital tendency to herniation.

The results in this class are good, possibly because of the above explanation, and this would seem to agree with the findings in the similar class in Group 1. There may be certain types of vaginal prolapse in which relaxation of the pelvic floor plays almost no part. One is certainly left with the impression that vaginal plastics will cure vaginal prolapse if there is no associated uterine prolapse.

The twenty-six cases summarized in Table 5 emphasize the relative size and importance of this group. The combined number of cases in Class B in both groups is 63.5 per cent. of the total sixty cases. It appears from a study of the previous classes that complete prolapse should be infrequent if operative relief is given early. Simple vaginal prolapse on the other hand often causes insufficient subjective symptoms to demand operative relief, or when patients with vaginal prolapse with sufficient symptoms do seek relief, we find that there is already a concomitant uterine prolapse. It is, therefore, in this class of patients, containing the majority of cases with relaxed pelvic floor, that most interest centers.

Of the twenty-six patients in this class, all but three were considered good surgical risks. One patient was unmarried, and three had had no pregnancies. The average age was 46 years. Symptoms dated back from one month to twenty-five years; but in only six cases had there been symptoms for less than one year. Six patients had been subjected to previous operations for the same complaint, and in two instances the operation had been repeated previous to coming to this clinic. In all these cases some form of perineal repair had been performed, and in four cases there had been, in addition, some form of abdominal operation. The chief complaints were "falling of the womb," backache, bearing down pains, incontinence of urine and in three cases actual appearance of the womb at the labia. It appears from these data that combined vaginal and uterine prolapse is more common in multiparous

TABLE 5.—GROUP 2, CLASS B, VAGINAL AND UTERINE PROLAPSE, TWENTY-SIX CASES

Case; Surgical No.	Married or Single; Age; No. of Children	Preoperative Condition	Previous Operation	Local Condition	Complicating Condition	Chief Complaint; Duration	Operative Procedure	Months to Final Observation	Result at Final Observation
1 10831	Married 41 2	Good	None	Large cystocele and rectocele, prolapse, fibroids	Myomas	Falling of womb and menorrhagia; 3 years	Anterior and posterior colporrhaphy, perineorrhaphy, supravaginal hysterectomy, incidental appendicectomy	20	Slight leukorrhea; slight cystocele and rectocele, firm perineum, slight descensus; occasional frequency and functional incontinence; coitus unsatisfactory; no life
2 11450	Widow 68 4	Good	Dilatation and curettage, perineal repair, 1914	Large cystocele, small rectocele, Slight prolapse	Urethral caruncle	Bulging from vagina, dysuria; 25 years	Anterior and posterior colporrhaphy, excision of caruncle	10	No relief of symptoms; subsequent operation for prolapse in September, 1920, within 1 year
3 5284	Married 47 5	Good	None	Moderate cystocele and rectocele, retroversion, slight prolapse	None	"Falling of womb"; 6 years	Anterior and posterior colporrhaphy, suspension, Moschcowitz	12	Dysuria; firm perineum; slight cystocele, no rectocele; uterus high, in good position
4 5280	Married 42 4	Good	Perineal repair 9 years ago, removal of left ovarian cyst 1 year ago	Moderate cystocele and rectocele, descensus	None	Backache; 1 year	Anterior and posterior colporrhaphy, ventral fixation, Moschcowitz; lysis of intestinal adhesions	52	Subsequent operation, hysterectomy because of abdominal pain (April 1, 1920, outside) stated there was no anatomic recurrence; excellent perineum, no cystocele or rectocele
5 9165	Married 48 5	Good	None	Large cystocele and small rectocele, descensus	None	Protrusion from vagina; 8 years	Anterior and posterior colporrhaphy, ventral fixation, incidental appendicectomy	24	No subjective symptoms; no cystocele or rectocele; uterus high and fixed; excellent result
6 4707	Married 58 None	Good	None	Large cystocele, retroversion, prolapse	None	Falling of womb, incontinence of urine; 3 months	Anterior colporrhaphy, ventral suspension	50	Frequency of micturition, uterus 1½ inches outside; complete recurrence
7 4528	Married 58 None	Good	None	Moderate cystocele and rectocele, retroversion, slight prolapse	None	Backache, falling of womb; 1 month	Anterior and posterior colporrhaphy	49	No cystocele or rectocele; cervix high, no symptoms
8 567	Married 55 3	Poorly nourished, senile	None	Large cystocele and rectocele, prolapse and retroversion	None	Falling of womb; 1 year	Anterior and posterior colporrhaphy, ventral suspension and fixation	84	Complete procidentia; backache
9 571	Married 43 9	Good	None	Small cystocele and rectocele, prolapse, retroversion	None	Falling of womb; 7 years	Anterior and posterior colporrhaphy, ventral suspension and fixation, dilatation and curettage, trachelorrhaphy	71	Cervix outside; backache; fair perineum

10 462	Married 42 5	Good	None	Large cystocele, small rectocele, prolapse, lacerated cervix	None	Falling of womb; 7 years	Anterior and posterior colporrhaphy, trachelorrhaphy, ventral suspension and fixation	84	Relieved; satisfied with result; anterior abdominal wall solid; cervix high and fixed; fundus anterior
11 2500	Married 33 5	Good	None	Small cystocele and rectocele, retroversion, prolapse, lacerated perineum	None	Abdominal pain; 18 months	Posterior colporrhaphy, ventral suspension	28	Recurrence of prolapse; pain and dragging sensation
12 2500	Married 40 3	Good	None	Moderate cystocele and rectocele, lacerated cervix. prolapse	None	Backache and dragging sensation; 8 years	Dilatation and curettage, trachelorrhaphy, ventral suspension (Gilliam), posterior colporrhaphy	27	No recurrence; good result
13 1236	Married 38 8	Good	None	Large cystocele and rectocele, lacerated cervix, prolapse	None	Falling of womb, lower abdominal pain; 8 months	Dilatation and curettage, trachelorrhaphy, posterior colporrhaphy, ventral suspension (Gilliam)	6	Plastics satisfactory; fundus good anterior position; letter two years later, recurrence
14 2000	Married 58 2	Good	Two plastics, 2 and 5 years ago; abdominal operation 2 years ago	Moderate cystocele and rectocele, prolapse	None	Bearing down pain; 19 years	Bisection operation with implantation in rectus sheath	30	Prolapse; symptomatic recurrence; marked cystocele; failure
15 5118	Widow 58 1	Good	None	Lacerated perineum, prolapse	None	Falling of womb; 20 years	Bisection operation with implantation in rectus sheath	51	Uterus well supported; no prolapse, no cystocele or rectocele; examination shows carcinoma of anus
16 3350	Single 58 None	Good	None	Prolapse	None	Dragging sensation, functional incontinence of urine; 9 months	Myomectomy, bisection of uterus with implantation in rectus sheath	56	Symptoms recurred; examination shows large cystocele, prolapse; fixation broken away
17 135	Married 68 8	Poorly nourished	Trachelorrhaphy and vaginal plastic 12 years ago	Large cystocele and rectocele, prolapse	None	Falling of womb; 22 years	Anterior and posterior colporrhaphy	85	No symptoms; large rectocele, some descensus
18 9078	Married 45 1	Good	None	Large rectocele, small cystocele, fibroids, retroversion	None	Uterus falls out; 21 years	Posterior colporrhaphy, supravaginal hysterectomy with suspension	18	Total relief; very slight bulging anteriorly and posteriorly; cervix fairly well supported
19 10140	Married 45 2	Fair	19 and 3 years ago abdominal operations for relief of prolapse, appendicitis and fibroid of uterus	Large cystocele and retrocele, retroversion, prolapse	Cholelithiasis	Falling of womb; 18 years	Dilatation and curettage, amputation of cervix, anterior and posterior colporrhaphy, bilateral salpingectomy, right oophorectomy, supravaginal hysterectomy with fixation; cholecystectomy	16	No cystocele or rectocele; cervix high, complete relief; (lipoma back); excellent result

TABLE 5.—Group 2, Class B, Vaginal and Uterine Prolapse, Twenty-Six Cases—(*Continued*)

Case; Surgical No.	Married or Single; Age; No. of Children	Preoperative Condition	Previous Operation	Local Condition	Complicating Condition	Chief Complaint; Duration	Operative Procedure	Months to Final Observation	Result at Final Observation
20 9007	Married 37 1	Poor	None	Large cystocele, small rectocele, lacerated cervix, retroversion, prolapse	Diabetes, heart disease (auricular fibrillation)	Backache; 3 years	Ventral suspension (Gilliam)	19	Uterus in good position, no cystocele or rectocele; abdominal wound satisfactory; dyspnea and fibrillation
21 10490	Married 48 7	Good	None	Large cystocele, small rectocele, prolapse	None	Falling of womb; 1 year	Trachelorrhaphy, anterior and posterior colporrhaphy, right salpingectomy, supravaginal hysterectomy, suspension of cervix, incidental appendicectomy	14	Dysuria, slight cystocele, firm perineum, fair support
22 4115	Married 38 8	Good	None	Moderate cystocele and rectocele, retroversion	None	Falling of womb; 2 years	Posterior colporrhaphy, ventral suspension	47	No symptoms; firm perineum
23 8879	Married 50 3	Good	None	Retroversion, some prolapse	None	Bearing down sensation; 6 years	Anterior and posterior colporrhaphy, ventral suspension (Gilliam), incidental appendicectomy	24	No cystocele or rectocele, no prolapse; dyspareunia; no other symptoms
24 6795	Married 66 9	Good	None	Retroversion, rectocele, moderate descensus	None	Frequent micturition, bulging from vagina; 1 year	Trachelorrhaphy, posterior colporrhaphy, supravaginal hysterectomy, bilateral salpingectomy, fixation of cervix and rectosigmoid	44	Excellent result, good perineum; no prolapse
25 11018	Widow 89 7	Good	Perineal and abdominal operation 14 years previously	Large cystocele, moderate rectocele, retroversion, moderate prolapse	None	Backache, protrusion cervix from vagina;	Dilatation and curettage, supravaginal hysterectomy	20	No symptomatic relief; frequency and dysuria; examination shows large cystocele with cervix coming to labia, lax perineum
26 6464	Married 36 4	Good	None	Cystocele and rectocele, moderate prolapse, lacerated cervix	None	Backache for 15 years, protrusion cervix	Anterior and posterior colporrhaphy, supravaginal hysterectomy, incidental appendicectomy	48	Complete symptomatic relief; examination shows small cystocele, thin perineum, cervix slight descensus

than nulliparous women, that its onset is insidious and that patients usually seek operative relief only when the lesion has progressed for years. It would appear that simple plastic operations, as evidenced by the number of cases with recurrence after such operations, do not always cure.

The operative procedures may be divided thus: (1) simple vaginal plastics; (2) abdominal operations, and (3) abdominal operation combined with vaginal plastics.

Vaginal plastics alone were done in Cases 2, 7 and 17 in which anterior and posterior colporrhaphies were performed. Of these, in Case 2 there was a complete recurrence, and the patient was reoperated on within a year. As this patient had already had one unsuccessful plastic operation a few years previously, it is obvious that an abdominal operation should have been performed. In Cases 7 and 17 there was no recurrence of symptoms, but when one patient (Case 17) was examined after a seven year interval, a large cystocele and some descensus were present. This patient, however, with her advanced age and hypertension, was not considered a good surgical risk at the time of operation. The good result after four years in Case 7 stands alone and may be more a symptomatic than an anatomic cure.

Abdominal operation alone was performed in Cases 14, 15, 16, 20 and 25. Bisection of the uterus and implantation above the rectus muscle was performed in Cases 14, 15 and 16, and in Case 20 a modified Gilliam suspension. Two patients, Cases 15 and 20, did well and at fifty-one months and nineteen months, respectively, after operation showed no recurrence anatomically or symptomatically. Cases 14, 15 and 25 recurred almost completely. A 60 per cent. failure is hardly satisfactory. The most marked feature of the recurrence was the great degree of vaginal prolapse, which suggests that had the abdominal operation been combined with vaginal plastics or by such a procedure as that of Moschcowitz to obliterate the posterior culdesac, the result would have been more satisfactory.

Combined abdominal and vaginal plastic operations were performed in seventeen instances, Cases 1, 3, 4, 5, 6, 8, 9, 10, 11, 12, 13, 18, 19, 21, 22, 23 and 26. In eleven cases both anterior and posterior colporrhaphy were performed while in the remainder, anterior or posterior colporrhaphy as indicated by the local condition.

There are seven patients in whom the simplest abdominal procedure, ventral suspension (Gilliam), was performed; Cases 6, 10, 11, 12, 13, 22 and 23. Of these seven patients, four, Cases 10, 12, 22 and 23, showed a good result at the final examination, with no prolapse. Three patients, Cases 6, 11 and 13, showed recurrence to the same or even greater degree than at the original observation. This is distinctly unsatisfactory and appears to condemn this type of operation

in women past the menopause. The mechanism by which recurrence occurs seems to be not only the failure of the round ligaments to maintain alone the position of the uterus but the fact that such a suspension does not protect the pouch of Douglas from the thrust from above. In fact, it appears to guide the intestines into the culdesac, thus tending to increase the amount of pressure brought to bear at this point and giving added strain on the ligaments, with the result that they stretch and thin out.

Ventral fixation of the uterus is the next procedure in simplicity. There were three cases in this group, Cases 5, 8 and 9. Examination showed one patient, Case 5, relieved, and two failures, with complete recurrence, Cases 8 and 9. There was no essential difference in these cases previous to operation, although one patient (Case 8) was generally senile and poorly nourished. The operation consisted of passing silk sutures into the fundus and over the rectus sheath, tying the knot intraperitoneally. The explanation of the failures in this small group seems to be much the same as when the modified Gilliam operation was performed, the weight of the intestine being guided into the posterior culdesac and exerting excessive strain, thus favoring herniation of the pouch of Douglas and pulling the uterus gradually away from the anterior abdominal wall. A retrospect of this group of combined operations in which some form of suspension or fixation was added to vaginal plastic operations shows poor results. Of the ten cases, five recurred completely. The explanations given above seem to explain the reasons for these recurrences and to lead to the conclusion that this type of operation should not be performed in women beyond the menopause or in whom for other reasons subsequent pregnancies need not be considered.

Two more procedures, the Moschcowitz and supravaginal hysterectomy with ventral fixation of the cervical stump, remain to be discussed. Only two patients returned for examination in whom the Moschcowitz operation, with ventral fixation of the fundus and posterior fixation of the cervix with obliteration of the pouch of Douglas, had been performed (Cases 3 and 4). Both of these patients showed entirely satisfactory results, twelve and fifty-two months after operation. There was slight dysuria in Case 3, and one patient (Case 4) had had a subsequent hysterectomy elsewhere for abdominal pain four years after operation. This patient (Case 4) was seen eighteen months after operation and showed an excellent anatomic result. Also she stated at the final examination that there had been no anatomic recurrence and that the subsequent operation was for pain only, very possibly from postoperative adhesions, which had not been entirely relieved by her operation in this hospital. This type of operation appears to be the most rational, as it creates a new and higher pelvic floor and can be used in young women contemplating subsequent pregnancies.

TABLE 6.—Group 2, Class C, Procidentia, Nine Cases

Case; Surgical No.	Married or Single; Age; No. of Children	Preoperative Condition	Previous Operation	Local Condition	Complicating Condition	Chief Complaint; Duration	Operative Procedure	Months to Final Observation	Result at Final Observation
1 5044	Married 55 4	Poorly nourished	Perineal repair 2 years ago	Complete procidentia	Myomas of uterus	Falling of womb; 20 years	Vaginal hysterectomy with fixation; anterior and posterior colporrhaphy	48	Slight cystocele, slight descensus vaginal vault; fair result
2 5580	Married 52 7	Good	Plastics six years ago	Complete procidentia	None	Falling of womb; 6 years	Bisection of uterus with implantation in rectus sheath	45	Marked rectocele; uterus well suspended; no prolapse
3 1721	Married 64 5	Good	None	Procidentia	None	Womb outside; 9 months	Vaginopexy and fixation of uterus	10	Complete recurrence
4 8916	Widow 45 3	Good	None	Complete procidentia	None	Falling of womb; 6 years	Dilatation and curettage, posterior colporrhaphy, ventral fixation	19	Slight cystocele, prolapse, postoperative hernia
5 8956	Married 45 6	Good	Right nephrectomy 2 years ago	Large cystocele and rectocele, procidentia	None	Falling of womb; 6 months	Anterior and posterior colporrhaphy, supravaginal hysterectomy, bilateral salpingectomy, left oophorectomy, incidental appendicectomy	24	Ventral hernia in scar; no cystocele or rectocele; cervix high
6 4495	Married 52 2	Good	None	Procidentia	None	Falling of womb, frequency of micturition; 8-10 years	Bisection of uterus with implantation in rectus sheath	47	Large cystocele; no prolapse; feels well
7 10075	Married 37 10	Poorly nourished	None	Small cystocele and rectocele, procidentia	None	Falling of womb for years, outside 6 months	Amputation of cervix, anterior and posterior colporrhaphy, supravaginal hysterectomy, bilateral salpingectomy, left oophorectomy, incidental appendicectomy	16	Complete relief; small cystocele, cervix attached to anterior abdominal wall but descends somewhat
8 9645	Married 46 7	Good	None	Procidentia	None	Falling of womb, outside; 13 years	Supravaginal hysterectomy, ventral suspension of stump, posterior colporrhaphy, incidental appendicectomy	19	Large cystocele; good perineum; no prolapse
9 7585	Married 47 2	Good	None	Procidentia	None	Weakness, protrusion from the vagina; 6½ years	Posterior colporrhaphy, supravaginal hysterectomy, bilateral salpingectomy, suspension of cervix	34	Rectocele; ventral hernia; cervix supported fairly high

In six cases (Cases 1, 18, 19, 21, 24 and 26), supravaginal hysterectomy was performed, with, in two cases (Cases 19 and 24,) ventral fixation of the cervical stump and in the others suspension of the stump by the round and broad ligaments. The supporting value of round ligament suspension of the cervical stump seems great in spite of the recent contention of Bissell.[12] On the whole, the results in these cases are good, with complete relief and anatomic cure in Cases 18, 19 and 24, only slight descensus and frequency in Cases 1 and 26 and a very slight bulging of the anterior vaginal wall in Case 21. This type of operation appears to have given better results than any other and to be a logical procedure if accompanied by vaginal plastics. To be sure, it does not create, as in the Moschcowitz operation, a new pelvic floor and protect the pouch of Douglas, but it elevates the culdesac and should securely fix that portion attached to the cervix.

A retrospect of the last eight cases shows satisfactory results. Both types of operation must be adapted to individual cases. It is suggested that the Moschcowitz procedure, obliterating the culdesac by posterior fixation of the cervix, might be applied after supravaginal hysterectomy. It is probable that when a marked herniation of the culdesac is demonstrable at operation, as described by Jones,[7] the Moschcowitz procedure is more suitable than hysterectomy.

The absolute influence that childbearing may have is not evident. The unmarried woman in this class presented an advanced lesion and it partially recurred after a radical operation, Case 16. Two patients (Cases 22 and 17), with eight children, did well after relatively simple procedures.

Table 6 summarizes the cases of procidentia or complete uterine prolapse. There are nine patients, varying in age from 27 to 64, all had children and the chief complaints were either "falling of the womb" or a description of the womb being outside. The symptoms had been present from six months to twenty years. All were good surgical risks.

The operative procedure varied widely. In Cases 1 and 2 previous vaginal plastics had been performed. In three cases, Cases 2, 3 and 6, no vaginal repair was performed as a part of the procedure in this clinic. In Cases 2 and 6, bisection of the uterus with implantation under the anterior rectus sheath was performed. In Case 3 the intra-abdominal procedure consisted of antiversion of the uterus, with vaginopexy and fixation of the uterus to the anterior abdominal wall. This resulted in complete recurrence in ten months. Presumably the sutures gave way. The reason for omitting a vaginal repair is not clear. In these three cases the vaginal prolapse recurred completely and is evidence that a combined operation is preferable. The cervix

12. Bissell, D.: Surg., Gynec. & Obst. **31**:578-583 (Dec.) 1920.

in both cases of bisection of the uterus remained well fixed to the abdominal wall; but as Frank [8] has shown, this does not prevent herniation of the pelvic floor.

In six of the nine cases a vaginal repair was performed at the same time as the abdominal operation. The most common abdominal procedure in this class was supravaginal hysterectomy with round ligament suspension of the cervical stump, Cases 5, 7, 8 and 9. As a group, these patients did well; two patients (Cases 5 and 7) were completely relieved. In Cases 8 and 9, respectively, a small cystocele and rectocele recurred; but in both cases the cervical stump remained high and the symptomatic relief was complete. It is unfortunate that no patients on whom supravaginal hysterectomy with ventral fixation of the stump was performed returned for observation, since this maneuver offers a little better chance for permanent relief. Graves [10] states that it is the operation of choice. It appears, however, that better vaginal repair might have given complete anatomic cure in Cases 8 and 9. In Case 8, in which a cystocele recurred, no anterior colporrhaphy had been performed. In Case 4, in addition to posterior colporrhaphy, ventral fixation alone was performed, resulting in recurrence of the uterine prolapse with cystocele. This is evidence again that simple suspension and fixation operations, rather than obviating the pressure in the pelvis, tend to increase it by guiding the bowels into the posterior culdesac. In Case 1 a vaginal hysterectomy, with suspension of the vaginal vaults by broad and round ligaments, was performed after the Mayo method. This gave a very fair result, with but slight cystocele, four years later.

The results in Class C bear a striking relation to the performance of a combined operation. In all cases in which there was a single procedure, that is, either an abdominal or a vaginal operation alone, there has been a recurrence. On the whole, supravaginal hysterectomy with round ligament suspension of the stump in conjunction with vaginal plastic operation appears to insure a good final result.

A summary of Group 2 patients shows that of the forty-two patients, those in Class A with simple vaginal prolapse (seven cases) were relieved by simple plastic operations on the vagina with one exception. The twenty-six patients in Class B upon whom several distinct types of intra-abdominal procedure were performed show a much less commendable end-result. The cases submitted to simple vaginal operations (three cases) showed complete recurrence in two cases; those submitted to abdominal operation alone (five cases) showed complete recurrence in three instances; those submitted to a combined vaginal and abdominal operation showed five recurrences in eighteen cases. The recurrences occurred in those patients in whom some form of simple ventral suspension or fixation of the uterus had been per-

formed. The results in the nine cases in Class C are no more encouraging since only five cases are satisfactory. All the uncombined operations (three cases) failed, and one ventral fixation with vaginal repair failed. In the whole group there are fifteen failures (35 per cent.) in forty-two cases.

A comparison of Groups 1 and 2 shows a great difference in end-results. The results in Group 1 are satisfactory whereas those in Group 2 are less good. The reason for this seems to depend on the more advanced lesions in Group 2, due to the progressive nature of this disability. The fact that patients do not seek operative relief until the anatomic lesion is very extensive explains the relative predominance of Group 2 patients.

TABLE 7.—Data Concerning Cases in Which Report Was Made by Letter

No. of Cases	Type of Operation	Result
Group 1:	Class A—Vaginal prolapse	
3	Vaginal plastic	1 relieved, 2 not relieved
	Class B—Vaginal and uterine prolapse	
3	Vaginal plastic	2 relieved, 1 not relieved
3	Vaginal plastic plus ventral suspension of uterus	2 relieved, 1 not relieved
1	Vaginal plastic plus supravaginal hysterectomy with round ligament suspension of cervix	1 relieved
	Class C—Procidentia	
1	Vaginal plastic plus supravaginal hysterectomy with round ligament suspension of cervix	1 relieved
11		
Group 2:	Class A—Vaginal prolapse	
6	Vaginal plastic	2 relieved, 4 not relieved
	Class B—Vaginal and uterine prolapse	
3	Vaginal plastic	2 relieved, 1 not relieved
5	Vaginal plastic plus ventral suspension or fixation of uterus	3 relieved, 2 not relieved
	Class C—Procidentia	
2	Vaginopexy	2 relieved
1	Bisection of uterus and implantation in rectus sheath	1 complete recurrence
1	Vaginal plastic plus ventral fixation of uterus	1 not relieved
18		
Total, 29 cases		

It appears that simple vaginal prolapse in either group is relieved by repair of the local lesion, that a combination of uterine and vaginal prolapse is more permanently relieved when treated early (Group 1), and by a combined operation, in which the abdominal procedure must be more than simple suspension or fixation of the uterus, and that procidentia requires a radical operation, preferably of some type that obliterates or elevates the pouch of Douglas.

B. THE PATIENTS WHO REPORTED BY LETTER

Table 7 demonstrates the character of cases in which report was made by letter, the operation performed and the end-results obtained. This table includes eleven cases in Group 1 and eighteen cases in Group 2. Seven patients in each group report symptomatic relief. This demon-

strates the greater difficulties in relieving Group 2 patients, either symptomatically or anatomically. A study of the operative procedures used indicates that patients on whom only local repair operations were performed in both Groups 1 and 2 did badly. This is contrary to the results observed in the examined cases. The five cases of procidentia, however, show results proportionate to the data obtained on patients actually examined and on whom hysterectomy was performed the results are good. Compared with the more accurate data collected in the cases of patients that returned for examination, the relative group percentages are as follows: Group 1, examined cases, 89 per cent. successes, letter cases 63.6 per cent.; Group 2, examined cases, 65 per cent. successes, letter cases 50 per cent. The unsatisfactory outcome in Class B patients in both groups seems to result from an inadequate operative procedure.

COMMENT

The material studied is representative in that it contains the varying degrees and types of relaxed pelvic floor. It consists of unselected cases from the local community. The patients' ages vary from the second to the seventh decade. The duration of the interval from operation to observation was sufficiently long to justify acceptance of the results as final.

The class of patients with uncomplicated vaginal prolapse treated by local plastic procedures shows surprisingly good results; better in the group of patients examined than in those reporting by letter. This interpretation of results was possibly influenced by rigid insistence on anatomic cures at the final observation. The reason why cases of this type do not go on to uterine prolapse is not clear; but it appears that there may be cases in which local trauma permits of a local herniation without actually destroying the supporting power of the pelvic floor. It is well recognized that cases with complete perineal tears frequently do not progress to uterine prolapse. Possibly the good results in this group will be partially vitiated with time. The results seem, however, to indicate that when uterine prolapse is to accompany vaginal prolapse it commences simultaneously with the vaginal lesion, and that if, therefore, patients at examination show no uterine prolapse, they can be permanently relieved by local vaginal operations. Retroversion may be considered as the initial stage of uterine prolapse.

The class of patients with uterine and vaginal prolapse (Class B) show less satisfactory results. Of the patients reporting for examination, those in Group 1 showed satisfactory results, those in Group 2 unsatisfactory results (ten recurrences in twenty-four cases). Among the patients reporting by letter in Group 1, two patients in seven were unrelieved and in Group 2, three patients in eight were unrelieved.

Certain of the failures can be explained by unwise choice of operative procedure. Thus, in patients in this class in whom a simple repair or an abdominal operation alone was performed, there was frequently recurrence. And when a combined operation was performed, but the abdominal procedure was a simple Gilliam or other suspension or fixation operation, there were several recurrences. A combined operation in which the abdominal procedure was either some form of hysterectomy or a Moschcowitz procedure proved satisfactory. It appears, therefore, that a combined local repair and abdominal operation should be performed when uterine and vaginal prolapse are present.

The type of abdominal procedure to be employed may depend on whether the patient desires more children It is recognized that the Gilliam operation and even ventral fixation of the fundus do not endanger subsequent pregnancies. It is also accepted, and these results seem to coincide with this view, that such operations do not give a high percentage of successes in uterine prolapse. The Moschcowitz procedure also permits of subsequent pregnancies, and these results as well as those of Clark [2] demonstrate its efficacy in this class of patients. In Group 2 when future childbearing is of no importance, a wider range of operations is available and the findings in this clinic agree with the opinion of Graves,[10] Tittel [13] and others that supravaginal hysterectomy, with either ligamentous suspension or ventral fixation of the stump, is most efficacious.

In procidentia cases, Class C, the results have been equally discouraging. Four of the nine patients who returned for examination had recurrences and two of the four patients replying by letter were unrelieved; six failures in thirteen cases (almost 50 per cent.). Here again uncombined operations resulted badly, patients on whom even such a radical abdominal procedure as bisection of the uterus returning for examination with most distressing vaginal prolapse. Patients on whom a hysterectomy, or the Moschcowitz procedure was performed, in combination with a vaginal repair, usually did well, and removal of the uterus either from above or by vagina after the Mayo procedure has proved satisfactory. The necessity of combined operations and radical abdominal interference is again demonstrated.

The type of operation is now and then further influenced by the condition of the patient. It must be remembered, however, that the combined operation need not be performed at the same sitting. They have frequently been performed separately, usually with an interval of from a week to ten days, but sometimes after some weeks, the patient returning for the second stage when thoroughly rested. The

13. Tittel, K.: Zentralbl. f. Gynäk. **44**:517 (May 15) 1920.

vaginal procedure is necessarily performed first. The task set is never a simple one and the patient must be made to understand this from the beginning.

CONCLUSIONS

Vaginal and uterine prolapse are the result of weakness in supporting structures, either congenital, traumatic or combined, in origin. The condition is essentially a herniation.

Vaginal prolapse when unaccompanied by any uterine prolapse can usually be cured by local vaginal repair procedures.

A combination of vaginal and uterine prolapse always requires a combined local repair and an abdominal operation. The type of abdominal procedure must depend somewhat on the desire for more children and on the condition of the patient. Uterine suspension and fixation operations are usually insufficient. Supravaginal hysterectomy with suspension or fixation of the cervical stump or the Moschowitz procedure has proved to be an efficient operation with lasting benefit

Procidentia must be treated by radical combined operations. Hysterectomy, either vaginal, or by the abdominal route, with some form of intra-abdominal vaginal support, and the Moschcowitz procedure have proved, in the cases studied, to give the best results.

SIXTEENTH REPORT OF PROGRESS IN ORTHOPEDIC SURGERY *

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CONGENITAL CONDITIONS

Steindler [1] describes twenty-five cases of congenital malformations and deformities of the extremities, and discusses their treatment by means of both original and classical methods. He finds in many cases an hereditary tendency and coexisting deformities. He believes that in the majority predevelopmental errors are the etiologic factors.

Torticollis.—Meyerding [2] has observed only twenty-six cases of congenital torticollis in nine years at the Mayo Clinic, and considers the deformity rather rare. In their treatment he has employed the generally accepted surgical method of dividing by open operation all tight structures and retaining the corrected position by plaster-of-Paris casts.

Fragilitas Ossium (Osteogenesis Imperfecta).—Frontali [3] reports a typical case of fragilitas ossium in which the thymus gland showed both atrophy and sclerosis. The other glands of internal secretion were normal. He maintains that examination of the thymus ought to be made in all cases of this type.

Congenital Dislocation of the Hip.—Calot [4] considers that recent progress in the treatment of congenital dislocation of the hip is due, first, to the discovery (?) that the true acetabulum in 90 per cent. of young children is formed by the ischial and not the iliac portion of the os innominatum; second, that the head and neck of the femur must be retained in the horizontal and transverse position throughout the whole treatment, the amount of rotation being exactly equal to the amount of torsion of the upper end of the femur, and the amount of abduction exactly equal to the angle of slope of the neck; and third, that the criterion of cure is the roentgen-ray evidence of a large and solid

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* This report is based on a review of 654 articles selected from about 875 titles having to do with surgery of the extremities and spinal column, appearing in medical publications, chiefly from July 1, 1920, to May 1, 1921. Only those papers seeming to represent progress in this branch of surgery have been selected for abstract and editorial comment.

1. Steindler: J. Orthop. Surg. **2**:639 (Dec.) 1920.
2. Meyerding: J. Orthop. Surg. **3**:91 (March) 1921.
3. Frontali: Riv. di clin. pediat., May, 1920.
4. Calot: Presse méd. **28**:666 (Sept. 22) 1920.

horizontal vault of bone, forming the roof of the original acetabulum. He states that this roof of bone can be observed to form during the time of fixation in the cast.

Galloway,[5] impressed by the bad results of his own attempts and those of others to cure congenital dislocation of the hip by bloodless methods of reduction, has been performing open operations in nearly all the cases which have come under his observation. He states that his operative findings have convinced him of the futility of obtaining a satisfactory replacement by manipulative methods in the great majority of cases. His conclusions are: 1. All cases should be treated by open operation and never by manipulation alone. 2. Open operation should be performed without first attempting to replace the head by manipulation. 3. The best age for open operation is between 2 and 3. 4. For children under 6, the anterior incision gives adequate exposure; in older or especially difficult cases the posterior incision should be employed. 5. In unilateral dislocations there is no upper age limit; although limited motion may result, the greater stability is more to be desired. 6. Never attempt to operate on both hips at one sitting. Galloway has performed fifty operations on thirty-six patients. He considers twelve cured. He records fourteen other "good" results, and six failures. Six are too recent for final report. He has had one fatality.

[ED. NOTE.—The recent findings of the commission appointed by the American Orthopedic Association, which will be given more fully in a future report, have shown that the cases of congenital dislocation of the hip which have resulted in apparently anatomically complete cures by the bloodless manipulative methods are very rare. They have not shown that the functional results are as discouraging as the roentgenologic findings might lead us to fear. Several clinics have demonstrated extensive series of cases in which the functional results were satisfactory in a majority of the cases. We are not prepared to accept Galloway's dicta on the basis of the present evidence, but in fairness to our own patients we must explain to them that the successful reduction of the dislocation may not mean a perfect anatomic cure, and that subsequent growth changes may occur. How serious an impairment of function these changes may represent later in life we cannot yet fully predict. Whether Galloway's open methods will give a larger proportion of good functional results and avoid the frequent deformations of the head, which we now know so often follow reduction by manipulative procedures, we do not yet know; but at the present moment we are by no means prepared to abandon the manipulative procedures and accept the open operation as a routine, as Galloway suggests.]

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5. Galloway: J. Orthop. Surg. **2**:390 (July) 1920.

Dubreuil-Chambardel [6] reports five cases of congenital dislocation of the hip occurring in children whose mothers had suffered from the same condition. The dislocations in the mothers had been corrected in early childhood.

Feutelais [7] reports a rare condition of congenital permanent contraction in flexion of both hip joints. There was no demonstrable central or peripheral nerve lesion. A firm fibrous band between the anterior superior spine and the thigh represented the tensor vaginae femoris muscle, and at operation both this muscle and the sartorius were found to contain no muscle tissue. These firm fibrous bands cast a heavy shadow in the roentgenogram. Resection of the restraining bands and retention in a plaster spica restored normal function.

Congenital Equinovarus.—Elmslie,[8] after describing very exactly the antomy of clubfoot and emphasizing the importance of a knowledge of its morbid anatomy as a prerequisite to treatment, expresses the opinion that tenotomies have been too freely performed, and that they often very definitely affect the ultimate function of the foot. Repeated manipulations and plaster casts are his methods of choice. Relapse, he states, is due to incomplete correction at first. The obstructions to be overcome are the astragaloscaphoid capsule, the displacement of the cuboid inward upon the os calcis, and the obliquity of the neck of the astragalus and anterior portion of the os calcis. He describes his operation, in which through an incision along the inner side of the foot the anterior two thirds of the internal lateral ligament, together with the astragaloscaphoid capsule, is completely cut away, thereby exposing the head of the astragalus, the tuberosity of the scaphoid, and the sustentaculum tali. From the outer side, the os calcis is cut through with an osteotome, one-half inch (1.3 cm.) behind its anterior border. The whole anterior part of the foot can now be abducted and elevated. If the obliquity of the neck of the astragalus is an obstacle, it can be similarly osteotomized.

Ober [9] previously has described a somewhat similar operation for resistant cases in which the patient is not less than 2 years of age. The skin incision is made from the posterior tibial groove three fourths of an inch (1.9 cm.) above the internal malleolus, extending forward across the mesial surface of the tibia, curving downward and ending at the inferior border of the scaphoid. This cuts the deep fascia, periosteum, annular ligament, and anterior portion of the deltoid ligament. The next step is removal of the astragalonavicular ligament from the scaphoid and clearing of the superior surface of the astragalus

6. Dubreuil-Chambardel: Bull. de l'Acad. de méd. **84**:281 (Nov. 30) 1920.
7. Feutelais: Rev. d'orthop., September, 1920.
8. Elmslie: J. Orthop. Surg. **2**:669 (Dec.) 1920.
9. Ober: J. Orthop. Surg. **2**:558 (Dec.) 1920.

of the anterior tarsal ligament, if it is adherent. The plantar fascia is divided subcutaneously. The adduction is corrected over a wedge, and forcible manipulation corrects the inversion and plantar flexion. The Achilles tendon is divided subcutaneously. Overcorrection must be possible with one finger. The first plaster is applied in only slight overcorrection because of the circulation, chiefly of the skin. Fourteen days later overcorrection can be greatly increased. Subsequent plasters are applied until full overcorrection is obtained, and are continued for four or five months. Then some form of clubfoot brace is worn for from six to eight months longer.

Summary.—1. The operation lengthens the foot. 2. Any desirable amount of overcorrection may be obtained. 3. No important vessels or nerves are injured. 4. The removal of the contractures allows one to obtain anatomic reposition of displaced bones.

Zadek [10] is insistent that the treatment of congenital clubfoot should be begun at the earliest possible moment. In all resistant cases, plaster-of-Paris casts should be used, and the treatment should be continued until the dorsum of the foot rests easily against the lower end of the leg. This postion, he thinks, should be maintained for about four months, in the average case. Rarely is a brace necessary. He believes that altogether too much reliance is being placed on braces, which are only retentive and not corrective. Frequent observation is necessary, and most of the relapses are due to insufficient treatment after the gross deformity has been corrected.

[ED. NOTE.—We have been more and more impressed, not only with the feasibility, but also with the completeness of functional cure, in quite extreme cases of congenital equinovarus by manipulative methods alone. The plaster technic must be meticulous and often prolonged, but we have yet to observe better end-results from any other method. Bone operations in children are surely to be discountenanced, except possibly the exact "skeletal adjustments" of Hoke. Small, awkward, and weak feet result. Neither the Ober nor the Elmslie operations should affect bone growth materially, and they may well prove entirely safe short cuts to function in resistant or neglected cases. We must, by this time, be familiar with the really disastrous results to future function for which the so-called Phelps operation is responsible.]

TUBERCULOSIS

Stigmas of Predisposition to Bone and Joint Tuberculosis.—Rivers,[11] reviews the extensive historical evidence of a tuberculous dyscrasia

10. Zadek, Isadore: Correction of Congenital Clubfoot in Infants, J. A. M. A. **75**:536 (Aug. 21) 1920.

11. Rivers: Brit. J. Child. Dis. **17**:59 (April-June) 1920; cont. **17**:140 (July-Sept.) 1920.

accumulated before the discovery of the bacillus. To this extensive and delightfully written review, Mr. Rivers, who is tuberculosis officer of the Barnsley District, West Ridings, Yorks, adds the data of his own experience. The three chief historical stigmas were red hair, icthyosis, and nasal abnormalities. From a study of over 350 cases of bone and joint tuberculosis and a series of normal controls, Rivers finds that red hair is about twice as common among tuberculous patients as among normal persons, icthyosis is also about twice as common, and nasal defects, chiefly catarrhal or atrophic rhinitis, are more than three times as common. Mental defects and hypertrichosis are also found much more frequently in tuberculous children.

General Treatment of Surgical Tuberculosis.—Sorrel,[12] the successor of Ménard as chief surgeon to the Municipal Hospital of Paris at Berck sur Mer, has become convinced that heliotherapy at the seashore is the most energetic treatment for bone, joint, and glandular tuberculosis. He places little value on drugs or auto-inoculation with fluid removed from tuberculous joints. The effects of serotherapy and tuberculin treatment he considers inconclusive. He is, however, convinced of the value of roentgen-ray treatment of glandular tuberculosis. In children, surgical intervention should rarely be undertaken, while in adults resection is the rule. After 50, he believes a cure from resection cannot be expected, and that amputation is generally indicated. His impression is that the actual duration of a tuberculous lesion is not shortened much by treatment, and that it completes a more or less definite cycle. We can, however, reduce the amount of bone destruction and allow healing to take place with the least disturbance of function.

Negri's[13] experience confirms the value of heliotherapy, even at comparatively low altitudes, and van Ree,[14] after observing the effects of roentgen-ray treatment in 470 cases of glandular tuberculosis, mostly in the neck, is enthusiastic over the brilliant results. Improvement occurred in every case, and 85 per cent. of the patients were clinically cured within a few months. The exposures are made at three-week intervals. Aspiration or occasionally the excision of a favorably located single gland may hasten the healing process. Abscesses should not be opened by incision. Deep bone processes do not seem to be so tractable, but superficial lesions of the smaller bones and joints often heal rapidly under roentgen-ray therapy, especially if the sequestrums are removed.

Steindler,[15] reviewing 350 cases of joint tuberculosis, of which 225 were treated conservatively, twenty-eight operatively, and ninety-seven

12. Sorrel: Presse méd. **29**:101 (Feb. 5) 1921.
13. Negri: Policlinico **27**:324 (Oct.) 1920.
14. Van Ree: Nederlandsch Tijdschr. v. Geneesk. **2**:1985 (Nov. 13) 1920.
15. Steindler: J. Iowa M. Soc. **11**:33 (Feb.) 1921.

not at all, feels that the strongest advocacy should be given to heliotherapy and outdoor life and, usually, to institutional care. All severe cases of spinal or hip joint disease should be treated in recumbency, and the general trend of his opinion is towards lengthening the period of recumbency.

Mixed Lung and Joint Tuberculosis.—Some rather interesting observations have been made by Jacquemin,[16] physician to a hospital to which these combined cases are sent. He thinks that he has discovered that the prognosis depends on which process is primary. He considers that the secondary process acts like a derivation or fixation abscess, and a lessening of the severity of the primary process may be expected. When the process in the lungs is secondary to the bone or joint process, the lungs soon become compromised; but when the bone or joint process has developed secondarily to a lung process, he has found it to do little harm, and he believes it should be looked on as a favorable sign. He believes that these secondary bone foci should not be operated on, citing disastrous examples among his cases. On the other hand, all primary bone or joint foci should be excised with the object of relieving what he considers a sort of sacrificial process in the lungs.

Tuberculosis of the Spine.—Calvé and Galland's [17] conception of the mechanics of Pott's disease is that of a fracture of the spine, the two segments balancing on the diseased articulation. In children, they believe the disease heals completely in from three to five years, with the minimum of deformity, by conservative methods employed in recumbency. In adults, after two years of recumbency, they believe a long, ankylosing bone graft makes safe a quicker return to function, relapses being very common without this added protection.

In adults, Calvé [18] prefers the Albee transplant in the lumbar region, but elsewhere the bone periosteal ankylosing operation. He believes that from two to three years of recumbency should precede the operative procedure, and six months of recumbency should follow the operation. A protective corset should be worn for two or three years after the patient is allowed to be up. Calvé and Galland consider that even in children it is possible that a short bone graft, limited to the diseased vertebrae, may immobilize the articulation more securely than Nature can alone. They insist, however, that the classic treatment of recumbency and an antituberculous regimen must be continued until repeated roentgenologic examinations demonstrate that a complete bony ankylosis has been secured. Calvé reports four cases in which he has attempted to relieve the paraplegia by inserting his special curved

16. Jacquemin: Bull. méd., Sept. 18, 1920.
17. Calvé and Galland: Rev. de chir. **58**:340, 1920.
18. Calvé: Médécine **2**:38 (Oct.) 1920.

trocar into the spinal canal through the intervertebral foramen, with the object of relieving the pressure from a suspected abscess within the canal. This method was reviewed and comment on it was made in the fifteenth report of progress. One of the four patients he now reports cured, two are improving, and in the fourth he considers the result a failure.

Stone [19] reviews sixty-five cases of caries of the vertebrae, in thirty-three of which operation was performed in one way or another to obtain ankylosis. The remaining cases received the classic nonoperative treatment. Nineteen per cent. of the patients treated without operation developed abscesses; 42 per cent. of the patients operated on developed abscesses. He states that operation did not control the progress of the deformity. The mortality of the operations was 15 per cent. He concludes on the basis of his review that operative procedures are not indicated in children and that after care must be continued for as long a time as in the cases in which operation was not performed.

[ED. NOTE.—It is evident that more general employment of roentgen-ray therapy is demanded, surely in all the more or less superficial forms of surgical tuberculosis. The wide experience of Stromeyer [20] that roentgen-ray treatment is of great value in young individuals, and as a postoperative treatment, is one of the latest bits of testimony to this effect. It is evident that heliotherapy is being gradually accepted as perhaps the most important part of an antituberculous regimen, quite irrespective of whether heliotherapy of the Alpine height type is or is not available. It is evident also that in spinal caries more and more reliance is being placed on prolonged recumbency to effect a permanent cure, and that less and less reliance, especially in children, is being placed on bone grafting operations. The recent report of the commission of the American Orthopedic Association on ankylosing operations on the spine gives added strength to this view. The report will be discussed more fully in a future report of progress.]

Operative Treatment of Healed Hip Disease.—Rogers and Peabody [21] believe that the three groups of these cases which they differentiate demand different operative procedures. In the first group, in which the lesion has healed with firm bony ankylosis, a curved osteotomy just below the trochanter, or transtrochanteric, serves to correct the adduction and flexion deformity. In the second group of cases, in which there is some remaining motion, but a fixed deformity, they

19. Stone: J. Missouri M. A. **17**:367 (Sept.) 1920.

20. Stromeyer: Deutsch. med. Wchnschr. **46**:514 (May 6) 1920; cont. **46**: 542 (May 13) 1920.

21. Rogers and Peabody: J. Orth. Surg. **2**:589 (Nov.) 1920.

advocate an arthrodesis of the joint, no matter how slight the motion. In the third group, in which there has been extensive destruction of the head and neck, and in which the trochanter is elevated and rests on the ilium, a simple arthrodesis in position may be less sure to succeed than taking off the top of the trochanter, cleaning out the old acetabulum, and placing the trochanter in it. The authors maintain that firm ankylosis of the joint in from 20 to 30 degrees of flexion and 10 degrees of abduction is the best functional result of tuberculous hip disease.

[Ed. Note.—We are in accord with the operative procedures which the authors advocate in the types of old hip disease. We are also ready to express our conviction that, in perhaps the majority of cases of tuberculous disease of the hip joint, occurring even before growth has ceased, bony ankylosis in the position they suggest is the best result to be obtained from our present methods of treatment. We are not convinced, however, that if recumbency were more generally employed, even in the mild cases, and some method (perhaps allowing a certain amount of painless voluntary motion) could be instituted for increasing the blood supply and decreasing the atrophy, useful motion in this peculiarly shaped joint could not be retained after the disease is overcome. We have observed an occasional result of this sort in true tuberculous disease of the hip, and although the range of motion has not been complete, and temporary joint strains (not lighting-up of the old process) have occurred, we believe these patients to be happier, less conspicuous, and more efficient than those with firm, painless ankylosis of the hip in a favorable position. We are not ready to give up striving to devise methods of cure of tuberculosis of the hip with useful motion remaining. We shall always have the recourse to ankylosis by arthrodesis if we fail.]

Spina Ventosa.—Rocher and Lasserre [22] have obtained rapid cures in nine cases of spina ventosa by making a lateral incision, scraping out the spongy tuberculous bony tissue beneath the periosteum, and suturing immediately. Continuous traction on the finger is maintained until healing and sufficient bone regeneration have occurred.

Arthrodesis of the Wrist.—Ely [23] has employed with satisfaction in tuberculosis of the wrist a tibial graft implanted in the radius, laid in a channel made in the carpal bones and terminating in the third metatarsal, the hand being maintained in slight dorsal flexion.

[Ed. Note.—Considering the experiences of Lexer and others with the lack of permanence of grafts which bridge articular surfaces, we should doubt the constant success of this procedure, unless the carpal

22. Rocher and Lasserre: J. de méd de Bordeaux **91**:466 (Sept. 10) 1920.

23. Ely, L. W.: An Operation for Tuberculosis of Wrist, J. A. M. A. **75**: 1707 (Dec. 18) 1920.

joints were destroyed as well. The experience has been that after a considerable period, a solution of continuity in the graft is very prone to occur where it crosses the articulation. There are several articulations in the wrist which must be thus crossed.]

Tuberculosis of the Knee Joint in Children.—Henderson [24] is convinced that the chief reliance in the relief of this condition must be open air, sunshine, tonics, good food, and general hygiene. His keynote is conservatism, with rest in the acute stage, and a Thomas knee splint during convalescence. Rarely is resection advisable.

Tuberculosis of the Foot.—Revel [25] believes that a thorough removal of tuberculous disease of the foot may be expected to eradicate the disease and afford better weight bearing than amputation and an artificial foot. He illustrates his article by three cases, one of thirty years' standing. Access to the disease is obtained by Delbet's method of cutting straight across the dorsum of the foot at the mediotarsal joint or between the tarsus and metatarsus, dividing all the dorsal tendons, nerves and vessels. The forefoot is swung down, and lengthwise incisions are made if necessary. All disease is dissected away and the forefoot is then sutured back in place, somewhat shortened, but still useful for weight bearing.

[ED. NOTE.—We have observed the result in one such case in an adult in whom for ten years no sign of the disease has recurred and the patient walks with a scarcely perceptible limp.]

RICKETS, OSTEOMALACIA, OSTEITIS FIBROSA AND OSTEITIS DEFORMANS

Rickets.—Nathan [26] summarizes some recent research which seems to substantiate the claims that rickets is a deficiency disease. Empiric observations had already demonstrated the efficiacy of cod liver oil in the treatment long before its high content of fat soluble vitamin was known. In any event, we should use it as a preventive and as a method of treatment, assuming for the present a deficiency factor which this supplies.

The work of McCollum, Simonds, and Parsons [27] in producing "rickets" and similar diseases in rats by deficient diet seems to bear out these conclusions. Rats were fed on diets made up from more than 300 formulas in which there was a deficiency of the fat soluble vitamin A or of calcium. The diets produce in young rats disturbances in the growth and formation of the skeleton. When the diets were supplemented by purified food additions to make good these deficiencies,

24. Henderson: Minnesota Med. **3**:463 (Oct.) 1920.
25. Revel: Rev. de chir. **58**:205 (March) 1920.
26. Nathan: Presse méd. **28**:577 (Aug. 21) 1920.
27. McCollum et al.: J. Biol. Chem. **45**:343 (Jan.) 1921.

normal nutrition and skeletal growth were induced. They conclude that it is only possible at present to state that the etiologic factor in this condition is to be found in an improper dietetic regimen.

Working with Shipley,[28] the same observers administered cod liver oil in carefully regulated amounts to rats in which experimental rickets had been produced by feeding on a diet deficient in calcium, sodium, and chlorin ions, as well as in fat soluble vitamin A. The animals were in such a condition that life could have lasted only a few days longer. The results showed ocular and seemingly conclusive evidence that there was some substance in the oil which caused calcium to be deposited in the same fashion as in the spontaneous healing of rachitis in man.

Phemister, Miller, and Bonar[29] have been studying the effect of phosphorus in rickets. After careful microscopic and roentgenologic observations they conclude that its method of action is little understood. In the case of cod liver oil, the result might be attributed to the presence of fat soluble A, but this could not be the explanation for the action of phosphorus. There is a trace of phosphorus in cod liver oil in organic combination, but it is improbable that it is present in sufficient quantity to exert any influence. Evidently phosphorus and also cod liver oil in some way restore the power of normal ossification, which in rickets is temporarily lost. They consider that their experiments have refuted the contention of Wegner, who believed that phosphorus was incapable of assisting in ossification in rickets until some other factor restored the altered calcium metabolism to normal.

[ED. NOTE.—Cod liver oil seems to be capable of bringing about normal bone formation in rickets. Phosphorus and actinotherapy seem capable also of so doing in certain instances. We may safely conclude that rickets is a disease of nutrition, but we apparently cannot yet positively say whether there is only one agent or whether there is more than one agent which can supply the deficiency in itself or make available substances necessary to normal bone formation and calcium deposition.]

Osteoclasis versus Osteotomy.—Blachard[30] considers the results from osteoclasis (Grattan), even in severe anterior bowlegs with the curvature near the joints, superior to the results from osteotomies. Nonunions following osteotomy are by no means rare, but he has never known one to occur after osteoclasis. In severe cases the Achilles tendon may be divided, and if, in an extreme bowing, the several osteoclases need to be performed at slightly different levels, three or four months

28. Shipley et al.: J. Biol. Chem. **45**:343 (Jan.) 1921.

29. Phemister, D. B.; Miller, E. M., and Bonar, B. E.: Effect of Phosphorus in Rickets, J. A. M. A. **76**:850 (March 26) 1921.

30. Blanchard: J. Orthop. Surg. **3**:1 (Jan.) 1921.

apart, there is no contraindication. Simple division of the Achilles tendon may be made at each operation. The other structures stretch sufficiently, and 10 cm. may be gained in length. Blanchard thinks that slow osteoclasis is dangerous. Not over eight seconds should be taken for the fracturing of the bone by the osteoclast.

Brüning,[31] on the other hand, advocates making several (up to six) osteotomies, with the Gigli saw, through small incisions. The traumatism is so slight that he does not hesitate to operate on both legs on the same day, even if the bones to be corrected are the femurs.

Actinotherapy in Rickets.—Riedel[32] reports from Ludloff's clinic excellent results in rickets from the use of the quartz lamp. We commented on the use of this form of actinotherapy in rickets in the fifteenth report of progress. He applies the light more vigorously than it is applied in Biesalski's clinic, using it daily, starting with three minutes and increasing three minutes a day up to thirty minutes. He states that delayed callous formation sometimes following osteotomy is greatly hastened by the light.

Rachitis Tarda.—Jean and E. Kollmayer[33] have observed 200 cases of rachitis tarda. They found these fell into three more or less distinct groups: (1) affecting chiefly the bones; (2) affecting the joints as well, especially the knees and feet; (3) affecting the alimentary canal with dyspepsia, diarrhea (often bloody), metrorrhagia, and bleeding from the bladder. Certain of the patients showed central nervous symptoms. They believe these changes to be best explained by external changes in the vascular system as a primary manifestation, and secondary changes in the endocrine glands. They think that the common cause of the osteomalacia and rachitis tarda is a decided lack of mineral salts in the food, owing to the insufficient fertilization of the soil during the war.

Osteomalacia.—Dieffenbach[34] contends that osteomalacia is a fairly common disease, though frequently unrecognized until spontaneous fractures call attention to the nature of the disease. The roentgenologic picture is fairly typical, but the clinical manifestations are typical also. There is a short, unsteady gait, with bent back and head, vague indefinite "rheumatic" pain referred to the different joints, and the presence of earthy phosphates in the urine. As the disease progresses, there is loss of height as the atrophic femurs or other long bones bend. In view of the almost epidemic character of the disease in central Europe, a nutritive disturbance would seem to be the probable etiologic factor, underfeeding, improper feeding, and deficiency in

31. Brüning: Deutsch. med. Wchnschr. **46**:1438 (Dec. 28) 1920.
32. Riedel: München. med. Wchnschr. **67**:838 (July 16) 1920.
33. Kollmayer, Jean, and Kollmayer, E.: Berl. klin. Wchnschr., Jan. 12, 1920.
34. Dieffenbach: Med. Rec. **97**:995 (June 12) 1920.

calcium, phosphorus, and the vitamins. There is also apparently a deficiency in suprarenal, parathyroid, and gonad secretion, perhaps primary, perhaps secondary. Treatment by rest, regulated diet, endocrine products, and actinotherapy, may be expected to bring about a cure in cases not too far advanced.

Cramer and Schiff [35] report the details of such a case of "famine osteomalacia" entirely relieved by nourishing food for a few weeks and the administration of calcium and phosphorus.

Chelmonski,[36] writing from Warsaw, describes a number of cases of what he calls alimentary osteoporosis encountered during the German occupation, when famine reigned. Recovery was prompt when the patient could have repose and proper food.

Freund and Lockwood,[37] in a case of osteomalacia, have studied the effects of certain endocrine extracts and oophorectomy on the metabolism of calcium and magnesium. During the administration of thyroid extract there was a marked increase in the loss of both calcium and magnesium. With pituitary extract there was a still greater loss of calcium, but little change in the magnesium content. During parathyroid administration there was retention of both calcium and magnesium. After removal of the ovaries there was a much greater loss of calcium and a slightly greater loss of magnesium than at any previous observation.

Ruggeri [38] reports a case of osteopsathyrosis in a child of 13, who had had twelve fractures. He found a deficit in the phosphorus, calcium, and magnesium balance. Under thymus extract administration this deficit was lessened and the condition improved, only to relapse later. There was associated a hypoplasia of the muscular and cardiovascular systems.

Hirsch [39] contends that rickets, osteomalacia, and osteoporosis belong to one great group of hunger osteopathies, and are differentiated only by the age and sex of the patients. Rickets is the disease of the child and the adolescent, osteomalacia that of the mature woman, and osteoporosis that of the age of involution. All these three affections showed a marked increase during and after the war. In all of them some influence of the endocrine system was noted.

Osteitis Fibrosa.—Langenskiöld [40] describes seven cases of osteitis fibrosa, giving the clinical and microscopic data. In localized lesions in which the bones become soft, resection of the abnormal bone is indicated. Diffuse lesions he considers hopeless.

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35. Cramer and Schiff: Rev. méd. de la Suisse Rom. **40**:746 (Nov.) 1920.
36. Chelmonski: Presse méd. **29**:115 (Feb. 9) 1921.
37. Freund and Lockwood: Ann. Med. **1**:67 (April) 1920.
38. Ruggeri: Pediatria **28**:953 (Oct. 15) 1920.
39. Hirsch: München. med. Wchnschr. **67**:1087 (Sept. 17) 1920.
40. Langenskiöld: Acta chir. Scandinav. **53**:1 (Aug.) 1920.

Greig [41] reports two cases of osteitis fibrosa, in one of which there was a history of severe muscle strain. In the other there were successive fractures of the leg and thigh. Following these fractures there was marked bowing, and the roentgenograms taken for the first time after the fractures showed an osteitis fibrosa. One questions whether the process antedated or followed the fractures. An osteotomy was performed to correct the deformity, with a good result.

Beust [42] has observed a case of congenital fracture of the tibia in which the examination showed evidence of osteitis fibrosa, with the formation of a bone cyst at the seat of fracture. He states that Stierlin has reported a congenital fracture of the tibia due to osteitis fibrosa. He raises the question whether osteitis fibrosa may not be a more frequent cause of congenital fractures than has been supposed.

Osteitis Deformans.—Romer [43] reports three cases of an osteitis resembling true Paget's disease, confined to a single bone and following a definite injury. In one case five, and in another twenty, years had elapsed since injury, and no other bones were involved. Pain was a more marked factor than in typical Paget's disease. The bones were operated on and the pain relieved. Normal bone growth occurred in the areas in which the bone was removed.

[ED. NOTE.—We have met one case of this sort in which no syphilitic taint existed.]

Sheath-Forming Osteitis.—Rénon and Géraudel [44] present roentgenograms of a patient having Marie's hypertrophic pulmonary osteoarthropathy. They think the term misleading, as only the shafts of the bones are involved. The photomicrographs show a double process in the bones, (1) the production of a sheath of spongy new bone, and (2) a calcification of the intercellular fibrillae and interfibrillar substances. The bones contain an excess of fat, while the calcium and magnesium content seems to be normal. Their own case was that of a man of 60, with cancer of the lung and a chronic pneumonia. In five similar cases on record the connection of the condition with disease of the lungs seemed certain.

SYPHILIS

Horwitz [45] has found the Wassermann reaction positive in only about 50 per cent. of the cases of bone and joint syphilis. Reliance must therefore be mainly placed on clinical findings. He recognizes three types: 1. Congenital, characterized by spontaneous fractures at or shortly after birth, epiphyseal involvement marked with joint symp-

41. Greig: Edinburgh M. J. **24**:324 (May) 1920.
42. Beust: Deutsch. Ztschr. f. Chir. **152**:60, 1920.
43. Romer: Lancet **1**:77 (Jan. 8) 1921.
44. Rénon and Géraudel: Presse méd. **28**:413 (June 23) 1920.
45. Horwitz: Am. J. Syphilis **4**:426 (July) 1920.

toms and symmetrical polyarthritis, though the joint motion is only slightly limited, and bone thickening, plus bone softening. 2. The late hereditary form. Here joint involvement is the exception and shaft involvement the rule, as shown by the saber shaped tibia, cortical thickening on the side of the convexity, and irregularity of outline. The tenderness is marked, but the disability is slight. Atrophy of the muscles in the lower extremity is common. 3. Acquired form. There is frequently synovitis, permanent or recurring. The degree of distention is out of proportion to the amount of pain and disability. The arthritis is usually secondary to shaft involvement and extension into the joint. The osteomyelitis begins as an osteitis extending into the medulla, and the symptoms are analogous to those of a pyogenic osteomyelitis, except that the new bone formation is more extensive. In the acquired form periosteitis is not as common as in the late hereditary type.

Gaenslen and Thalhimer [46] report a case of what they believe to be a syphilitic epiphysitis in an adolescent boy of 13. They suggest that the lesion is often mistaken for tuberculosis. They would differentiate it by (1) the predominance of cartilage, and the "riotous" appearance of the osteogenesis; (2) the lack of completion of the new bone formation; (3) the excess of calcium deposit in the cartilage in apposition to the areas of new bone formation; (4) the obliterating endarteritis in the thickened periosteum.

Cotton [47] has been impressed by the improvement which he has noted in five cases of Charcot joints from antispecific treatment. He thinks it stops further progress, and that actual bony repair has taken place in certain instances. The mechanical damage which may remain can be greatly helped by apparatus.

SCOLIOSIS

Schlesinger [48] has examined 12,860 schoolchildren and found that approximately 12.5 per cent. show curvature of the spine. The great majority of these are made up of what he terms constitutional curvatures and rachitic curvatures, the former being almost six times as common as the latter. The differential diagnosis between the two, he thinks, is easy, the constitutional being characterized by a long trunk, often graceful and quite flexible, while the rachitic shows a short, clumsy spine and a thorax markedly limited in mobility. The rachitic curves are much more common in the lower grade schools, and at the age of puberty. The etiology of the constitutional form is not clear to him, though he admits the probability of a constitutional anomaly of

46. Gaenslen and Thalhimer: J. Orthop. Surg **3**:8 (Jan.) 1921.
47. Cotton: Ann. Surg. **7**:488 (Oct.) 1920.
48. Schlesinger: Arch. f. Kinderh. **68**:289, 1920.

muscles, ligaments, or bones. Faulty posture alone at school, or outside, he thinks does not explain it. These children as a class are neuropathic, with pallor of face, irritability, and frequent enlargement of the thyroid gland. There is a large percentage of short sightedness among them, which he thinks may well be a predisposing factor. Schlesinger has found that the rachitic curve tends to grow worse and needs radical orthopedic treatment. The constitutional curve often grows better under rest and careful postural education.

Kleinberg,[49] in a long article, gives the details of the treatment of scoliosis which he employs since his abandonment of the Abbott method, which failed to cure scoliosis in his hands. His conclusions are: (1) The most effective means of recognizing scoliosis is by the frequent routine examination of the naked backs of all schoolchildren. (2) Corrective treatment should be instituted as soon as the curvature is discovered. (3) Treatment should be continued uninterruptedly for years, until such improvement has been obtained as will reasonably assure the arrest of the deformity. (4) Extension of the spine is the best form of treatment, arresting the progress of the deformity and in many instances reducing the deformity. It is the least uncomfortable and does not cause malformation of the chest. (5) When the jackets are changed there must be no opportunity for relapse of the deformity.

Estes,[50] working at Lehigh University, concludes that "functional scoliosis" occurs in from 10 to 20 per cent. of college men, 70 per cent. of the curvatures being left-sided. He believes the commonest causes are "flatfoot," occupational or developmental peculiarities, and the shortening of one lower extremity.

[Ed. Note.—The problem of scoliosis is ever with us, and the satisfactory explanation of its etiology is still lacking in the majority of cases. We may call it "constitutional," idiopathic, functional, developmental, or whatever we choose, but these terms do not explain. It is clear, however, that posture alone does not cause it, nor short legs alone, nor vertebral anomalies alone. Rickets, we know, is associated in certain younger patients in an apparently causal relation. We believe that we must assume some condition of the bones of a similar nature to that in rickets, before a poor posture, or a short leg, or a vertebral abnormality can act as an exciting cause. We seem to be able to correct the condition in certain cases of mild, flexible curvature; we seem to be able to arrest, perhaps improve the condition in certain cases of considerable severity. It has not been yet proved beyond cavil that we can untwist and unbend, much less overcorrect, the curvature

49. Kleinberg: Surg., Gynec. & Obst. **32**:364 (April) 1921.

50. Estes, W. L.: Causes and Occurrence of Functional Scoliosis in College Men, J. A. M. A. **75**:1411 (Nov. 20) 1920.

in cases in which there is a fixed rotary curve, without danger to the general health and the possible production of other deformities out of all proportion to the advantage of the correction of the curvature.]

FOOT STRAIN AND FOOT DEFORMITIES

Objective Signs of Foot Strain.—Freiberg [51] believes that pronation and abduction of the foot cause most strain at the tibiocalcaneal portion of the internal lateral ligament. Tenderness at the attachment of this ligament to the sustentaculum tali is a very characteristic sign. Freiberg suggests that an estimation of the amount of pressure at this point which causes pain may be a helpful sign in diagnosis of foot strain. In persons suffering from foot strain, pain is caused, as a rule, by pressure of less than 2½ pounds (1.13 kg.), most frequently by pressure of from ½ to 1½ pounds (227 to 681 gm.). If pain is caused by these light pressures, he interpretes it as a potential weakness in adductors and supinators.

Foot Strain in the Army.—Mebane [52] discusses foot abnormalities and their management in the light of his army experience in handling foot cases in base hospitals and development battalions, and on examining boards. The army considered that a normal foot must be unrestricted in its motion and must have its line of transmission of the body weight pass through the forefoot. The causes of foot trouble in the recruits were congenital, traumatic, and infectious. Success in treatment depended on accurate diagnosis, mechanical correction of abnormal positions, and the strengthening of weakened foot structures by means of carefully graduated exercises. Appropriate alterations in the tread of the Munson last army shoe and adhesive plaster strappings were employed. Felt pads and metal plates were found impractical in the army.

End-Results of Operations for the Relief of Hallux Valgus.—Spiers [53] has studied the end-results in ninety-six patients operated on in the orthopedic clinic of the Massachusetts General Hospital. The method generally employed until the last few years was excision of the head of the first metatarsal bone. There were a few cases each of cuneiform osteotomy of the first metatarsal, resection of the exostosis alone, and of the operation devised by Col. William Keller of the U. S. Army. This consists of the removal of the superior and mesial portion of the head, including the exostosis, and of the excision of a button of bone from the proximal end of the proximal phalanx. Spiers

51. Freiberg, A. H.: Objective Symptomatology of Footstrain, J. A. M. A. **75**:466 (Aug. 14) 1920.

52. Mebane: Mil. Surgeon **47**:428 (Oct.) 1920.

53. Spiers, H. W.: End-Result of Hallux Valgus Operations, J. A. M. A. **75**:306 (July 31) 1920.

found that (1) 66 per cent. of the excisions of the head were satisfactory to the patients; (2) by the excision method favorable results depended to no small extent on after-treatment; (3) the method was usually satisfactory in those patients whose occupations were sedentary; (4) after excision of the head, loss of normal range of motion is the rule; (5) in the unsatisfactory cases following excisions, the anterior arch was the trouble maker; (6) special and more or less permanent arch supports are essential after the head has been excised; (7) bony overgrowths frequently interfere with a satisfactory result after excision of the head; (8) the simple removal of the exostosis is rarely justified by the results; (9) the Keller method seems to offer a distinct advance in the operative treatment of hallux valgus.

Injuries to the Sesamoid Bones of the Great Toe.—Freiberg,[54] writing on this subject, recognizes the fact that divided sesamoids may be congenitally present in both feet and in both sesamoids, although the mesial sesamoid most often shows this abnormality. Injury to the sesamoids, including true fracture and subluxation, may occur. In none of his cases, except in a case of subluxation, was operation necessary to relieve the pain. The frequently renewed application of a thick felt pad, just behind the bone, until all tenderness had disappeared, and the wearing, for some time after this, of a transverse cleat, from one fourth to three eighths of an inch (0.6 to 0.9 cm.) in thickness, across the sole of the shoe, just behind the sesamoid bones, accomplished a permanent relief. Exercises for developing the power of the toe-flexing muscles completed the cure.

Hammond [55] reports two cases in which he found the mesial sesamoid adherent to the head of the first metatarsal bone. On removal, the sesamoids showed a tiny necrotic spot at this point.

Operations for Cure of Weak Feet.—Brodnitz [56] has modified Mueller's operation for the relief of severe flatfoot thus: Instead of reinserting the entire anterior tibial tendon into the scaphoid, he splits it and pulls one half through a hole in the scaphoid, and simply shortens the other half, thereby gaining a broader area of attachment, which he considers a great advantage.

Pingree [57] lengthens the tendons which are short and hold the foot everted, and shortens the adductors.

Human Prehallux.—Monahan [58] believes that the human prehallux, a structure growing from the inner border of the scaphoid downward and backward, is much more common than is generally recognized. He

54. Freiberg: J. Orthop. Surg. **2**:453 (Aug.) 1920.
55. Hammond: J. Orthop. Surg. **2**:506 (Sept.) 1920.
56. Brodnitz: München. med. Wchnschr. **40**:1158, 1920.
57. Pingree: J. Maine M. A. **10**:329 (June) 1920.
58. Monahan: Am. J. M. Sc. **160**:708 (Nov.) 1920.

has found it in 10 per cent. of cases of painful feet, many of them erroneously diagnosed as gout, rheumatism, and "flatfoot." It is not apparently a congenital structure in the human foot, but appears usually between the twentieth and fortieth years. Its origin is from some dormant ossification structure, often associated with syphilis or tuberculosis.

POSTURE

The Reaction of Bodily Mechanics to Cyclic Vomiting and Other Obscure Intestinal Conditions.—Talbot and Brown,[59] studying the effects of posture on the bodily functions of adults and children, have become convinced that vicious habitual postures inhibit normal intestinal function and induce constipation. Certain cases of cyclic vomiting, supposed generally to be due to some fault of innervation, have yielded rather strikingly to correction of the concomitant vicious posture by means of exercises and braces.

[Ed. Note.—We must not assume that all children with prominent abdomens, hyperlordosis, and feet which appear flat need a brace or even special exercises. Many apparently normal children go through this stage on the way to the erect posture of adult life. We may, however, recognize early the so-called congenital visceroptotic child, and associate causally obscure intestinal derangements, failure to gain weight normally, and general asthenia with this faulty position of the viscera and the accompanying faulty posture. The general improvement which takes place in their condition under postural treatment, when different methods of feeding, etc., have failed, is so extremely common as to be generally expected.]

Gastroptosis.—Beastrup [60] expects to find the stomach very low in women with long flat trunks, the upper abdomen curving in, and the lower abdomen prominent. The symptoms of dull headache, scanty frequent micturition, and constipation are usually present. He cites a case in which, after prolonged and unavailing medical treatment, a surprising gain in weight and a disappearance of symptoms occurred under massage and arsenic therapy, and through elevation of the foot of the bed, followed by the use of a supporting belt when the patient was allowed up.

Lee and Brown [61] are not prepared to say that many ailments of the nervous and gastro-intestinal system are caused by bad mechanical use of the body, but they recognize the frequent association of such symptoms. For example, their investigations have seemed to confirm

59. Talbot, F. B., and Brown, L. T.: Bodily Mechanics, Its Relation to Cyclic Vomiting and Other Obscure Intestinal Conditions, Am. J. Dis. Child. **20**:168 (Sept.) 1920.

60. Beastrup: Ugesk. f. Læger (Aug. 5) 1920.

61. Lee and Brown: Am. J. M. Sc. **160**:651 (Nov.) 1920.

the fact that certain cases of albuminuria in young men do not represent a true nephritis, but are associated almost exclusively with bad mechanical use of the body. In certain cases, such as poliomyelitis, one must be satisfied by bringing about a compensation in the same way as with an organic heart lesion. In other cases, a complete correction may be brought about by restoring proper bodily mechanics.

Reijs [62] has noted the effect which the attitude of the head and of the cervical spine has on the tonus of the fingers. This is a reflex action which can be demonstrated in decerebrated animals.

ARTHRITIS

The Nature of Arthritis.—In trying to construct a working hypothesis as to the cause of arthritis, Pemberton [63] considers it safe and certainly more reasonable to assume that a variety of factors may induce the substratum. These factors may be various types of infection, exposure to wet and cold, chronic intestinal conditions of which we have only imperfect knowledge, and the even more obscure internal glandular disturbances. The substratum is partly illustrated by a lowered sugar tolerance, which has been shown to accompany this disease almost invariably.

[Ed. Note.—We have in an earlier report of progress commented on Pemberton's work, which we believe is of exceptional merit. His method of attack seems most likely to succeed, and we find ourselves in heartiest accord with his belief that, considering the data now in hand, it is in the domain of physiology that the explanation of this disease is apparently to be found.]

Classification of Arthritis.—Pringle [64] considers that the differentiations and classifications of chronic arthritis suggested by Charcot, Goldthwait, and others, are unsound from a pathologic and etiologic, and even from a clinical aspect. Clinically, he recognizes two types of chronic arthritis: those which lead to ankylosis, both fibrous and osseous, and those which lead to ankylosis by deformity or locking. Pathologically, there are also two types, the inflammations and the degenerations. Between these, there are all gradations, and frequently both types are combined. He is, in general, in accord with Nathan's conclusions, and he quotes him as saying that, although a tooth or tonsil may be the portal of entry, when the micro-organism has once entered the blood stream, its connection with the portal of entry ceases. If a focus exists finally in the joint structures, it not only may cause local damage, but it also may sow general infection and metastases.

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62. Reijs: Nederlandsh Tijdschr. v. Geneesk. **2**:2883 (Dec. 25) 1920.

63. Pemberton, Ralph: Nature of Arthritis and Rheumatoid Conditions, J. A. M. A. **75**:1759 (Dec. 25) 1920.

64. Pringle: Lancet **1**:1106 (May 22) 1920.

[ED. NOTE.—Pringle, as a matter of fact, recognizes the two great types as does Goldthwait, and he is in still closer accord with the classification of Nichols and Richardson. Nearly all observers recognize the type called by Goldthwait the infectious, and sometimes the atrophic, type, and designated by Nichols the proliferative or ankylosing type, which, unchecked, leads to fibrous or true osseous ankylosis. Pathologically, this is the inflammatory type; etiologically, the infectious or toxic type. There is also general recognition of the second great type, the hypertrophic of Goldthwait, and the degenerative or nonankylosing type of Nichols and Richardson. Pringle says that this type leads to ankylosis by deformity and locking. The joint surfaces become irregular and may intermesh to such an extent that they become somewhat locked. They may become more or less fixed in positions of deformity, but we believe that they almost never become ankylosed, except occasionally in the spine, and that they are always associated with a certain amount of bony overgrowth, which made Goldthwait speak of them as hypertrophic. We think Pringle is wrong when he says that these two great types are frequently combined. The clinical pictures are sometimes confusing, and occasionally, in a case in which the fires of an ankylosing or infectious arthritis have burnt out, the patient may, in later life, develop Heberden's nodes or a morbus coxae senilis. By the same token, a case of degenerative arthritis may, while this form is still slowly progressing, have some focal infection which will cause the other type to become active. We still believe, however, that the two types are, clinically, entities and separate diseases.]

Group Study of Three Hundred Cases of Arthritis.—Harding [65] has studied a group of 300 cases of arthritis in a U. S. Army hospital, at Camp Lewis, Washington. In 89 per cent. of the cases some infectious process other than that in the joints themselves was found and treated; but Harding is not at all convinced that the particular germ present in the tooth or tonsil is necessarily the sole causative factor. The blood cultures were all negative, and 90 per cent. of the men had had an attack earlier in life. Harding thinks this finding calls for a most thorough and painstaking removal of all sources of infection in the arthritides of childhood, in order to avoid the attacks of later life. The routine treatment was rest, free catharsis, and the splinting of painful joints. The results of the injections of nonspecific proteins were unfavorable in the seventeen cases in which they were tried.

The Colon and Other Foci of Infection in Chronic Arthritis.—Bassler [66] reports forty-five cases of polyarthritis. In thirty-five, focal infections were found and removed, and in ten no focal infections

65. Harding: California State J. M. **19**:26 (Jan.) 1921.
66. Bassler: Am. J. M. Sc. **160**:351 (Sept.) 1920.

could be discovered. None of the patients received any apparent benefit for their joint condition. Careful examinations of the blood and stools were made. The sedimentary bacterial growths were grown anaerobically and aerobically with animal inoculations. Forty-four of the cases showed instances of chronic intestinal toxemias, thirty-four saccharobutyric in type, two indolic, and eight mixed. The patients were then treated by diets suitable to their toxemias and by vaccines, for at least five months, with directions to continue the diet for a year longer. Bassler thinks that there is no possibility of a cure in the true sense, but there is a possibility of definite benefit. There is good reason to believe that if attention is given to the colon as well as to other foci of infection early in the disease, many patients will be saved the after effects of chronic joint deformity and invalidism.

Newcomet [67] suggests that chronic intestinal disease offers an explanation for many cases of arthritis of the spine, especially in middle aged persons. He cites Pemberton's work as also suggesting this causal relation.

Chapman [68] reports twenty-one personally observed cases of chronic arthritis in which distant foci of infection were removed; 76.2 per cent. of the patients showed improvement, 19 per cent. showed no change, 4.8 per cent. were worse after treatment. The most striking effects were obtained in the genito-urinary cases, though the treatment was long and continuous. Rapid recovery was the rule in the cases in which the teeth were the focus of infection.

Bacterial Studies in Arthritis.—McMeans [69] has isolated a single strain of *Streptococcus viridans* from the pus of a submaxillary abscess in a patient free from joint symptoms. In his studies of this organism, he discovered that it was of low pathogenicity and had a rather high invasive quality, possessing the ability to attack joints of rabbits and produce a chronic suppurative arthritis. This tendency to attack joints was not lost after a period of three months of artificial cultivation.

Richards [70] has taken blood cultures in 104 cases of chronic arthritis in which syphilitic and Neisser infections could be ruled out. *Streptococcus viridans* was found in fourteen cases. Joint and periarticular cultures were taken during an acute exacerbation of the disease in forty-four cases, and *Streptococcus viridans* was found in four cases. Streptococci were found in the feces, by the Gram stain, in forty-two cases and were isolated and identified as *S. viridans* in five cases. Complement fixation tests with *S. viridans* were positive in sixty-eight cases.

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67. Newcomet, W. S.: Arthritic Changes in Spine: Their Relation to Roentgenologic Study of Gastro-Intestinal Tract, J. A. M. A. **75**:1418 (Nov. 20) 1920.
68. Chapman: Ann. Surg. **71**:648 (May) 1920.
69. McMeans: Am. J. M. Sc. **160**:417 (Sept.) 1920.
70. Richards: J. Bacteriol. **5**:511 (Sept.) 1920.

No foci of *S. viridans* could be detected in twenty cases, but of these negative cases, eleven showed a positive fixation test. In seven out of nine cases in which a streptococcus was found in the stool by the Gram stain, but could not be identified, a positive complement fixation test with *Streptococcus viridans* was obtained.

Nonspecific Protein Therapy in Arthritis.—Cowie [71] has been favorably impressed with the results of intravenous injections of foreign proteins in arthritis. He believes they should be used in those cases in which failure to secure benefit from the removal of foci of infection has occurred, or in conjunction with attempts to remove the focus. Unquestioned relief from pain often follows protein therapy, even though the focus is not removed; and, in addition to the improvement in the joint condition, the focus itself ceases to be active. As far as we know, foreign protein acts by combating infection. Acute and subacute processes that have not progressed beyond the first year, without structural change, are the ones which give the best results. Next to these are the cases which may show some structural change, but no ankylosis. Typhoid vaccine or a standard stock vaccine may be used. From one to ten injections, given at intervals of twenty-four or forty-eight hours, will demonstrate whether benefit is to be received. No anaphylactic phenomena accompany these injections, even though a second course of treatment may be instituted after an interval of several months.

Second Great Type of Arthritis.—Ely [72] includes under this term osteo-arthritis, arthritis deformans, hypertrophic arthritis, degenerative arthritis, and metabolic arthritis. He considers that this form has two distinguishing clinical features: (a) bone production (lipping or spurring) at the joint line, and (b) absence, except in spinal involvement, of union between the bone ends. Its main pathologic feature is the presence of an aseptic necrosis in the bone near the articular surface. This he thinks is the primary change, and the changes in the cartilage and the bony proliferation follow this. Ely thinks that the primary cause, in the great majority of cases, is infection in the alveolar processes of the jaw. Trauma is effective only as an added insult to a joint already diseased. This is called senile arthritis merely because elderly people are more prone to it, as they are to alveolar infections.

[Ed. Note.—We wish we could be as convinced as Ely seems to be that alveolar infections represent the usual important etiologic factor. His report of cases is impressive and his microscopic study very valuable, but we have not been so successful as he in finding foci in many of these cases.]

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71. Cowie, D. M.: Nonspecific Protein Therapy in Arthritis, J. A. M. A. **76**:310 (Jan. 29) 1921.

72. Ely, L. W.: Second Great Type of Chronic Arthritis, Arch. Surg. **1**: 158 (July) 1920.

Tubby,[73] writing on senile painful hip, states that there is a focus of toxemia in the jaws in 75 per cent. of the cases. Intestinal, gall-bladder, tonsillar or middle ear foci are found in the remainder. He believes that a direct or indirect trauma to the joint results in the localization in the hip. His treatment, after the foci have been removed, is to relieve intra-articular pressure by recumbency for from four to six weeks, with traction on the limb. Every day the hip is moved passively five or six times. When the patient is allowed to be up, he applies a special caliper splint, with a pelvic girdle and a drop catch at the knee. For cases not thus relieved he advises excision of the head of the bone, with section of the adductors, to allow the limb to be immobilized in complete abduction.

Gentian Violet in the Treatment of Purulent Arthritis.—Churchman[74] believes that pyogenic arthritis due to *Staphylococcus aureus* or to the pneumococcus may be cured with the retention of joint mobility, by his method of joint lavage and staining with gentian violet. Whether the joint in this form of infection should ever be opened, unless the infection has become largely extra-articular, he doubts. Churchman is a little more guarded in his statements concerning gonorrheal arthritis, but he believes that the clinical course of the cases treated warrants the belief that lavage and staining offer a method of controlling the disease.

Acute Arthritis in Infants.—Johnson,[75] studying seventy-three cases of acute arthritis in infants, concludes that it is not a very rare condition, but serious in nature and usually secondary to an infection of the epiphysis. It may spread to the periarticular tissues without affecting the joint proper. Clinically, there is marked swelling, tenderness, redness and induration. Streptococcal and gonococcal infections are usually polyarticular; pneumococcal infections commonly affect only one or two joints. Fifty-three per cent. of his patients died, 9 per cent. were discharged improved, 38 per cent. were followed up and later reported well or greatly improved. The older the baby, the better the chance of recovery.

PARALYSIS, ANTERIOR POLIOMYELITIS, SPASTIC PARAPLEGIA, ETC.

Family Outbreaks of Epidemic Poliomyelitis.—Mulsow and Matousek[76] state that in all the widespread epidemics of poliomyelitis the occurrence of several cases in one family, has been recorded in a

73. Tubby: Practitioner **105**:325 (Nov.) 1920.

74. Churchman, J. W.: Gentian Violet in Treatment of Purulent Arthritis, J. A. M. A. **75**:583 (Aug. 28) 1920.

75. Johnson, F. E.: Acute Arthritis in Infants, Am. J. Dis. Child. **21**:170 (Feb.) 1921.

76. Mulsow, F. W., and Matousek, W. J.: Family Outbreaks of Poliomyelitis, J. A. M. A. **76**:159 (Jan. 15) 1921.

number of instances, although it is rare. Most often the disease has developed simultaneously in the different children. In one family of eleven children, there were four cases, two of the girls being affected at one time and two of the boys two weeks later. No source of infection was discovered.

[ED. NOTE.—This evidence, as far as it goes, suggests that the infection is not usually from contact of one child with another, but simultaneous exposure from human or other contact. And if not from one child to another, is it not more probable that other than human carriers are the common sources of infection? In the exanthemas, human contact contagion is so often and so positively suggested that the burden of proof must be on those who suspect a nonhuman carrier. In poliomyelitis, the evidence of human contact contagion is so doubtful and rare that the burden of proof seems to be on those who maintain that the human carrier is the common source of infection.]

Treatment of Poliomyelitis.—Saethre [77] emphasizes the importance of preventing deformity from the very earliest stage of the disease. At first, perhaps with the help of simple apparatus, all the muscles must be maintained in a completely relaxed condition, a passive repose. Especially important is it to avoid the over-stretching of the paralyzed muscles. No exercise until all pain, hyperesthesia, and Kernig's sign have disappeared.

Mackay [78] maintains that every child surviving the acute stage of poliomyelitis can be assisted to regain muscle power. The cardinal points in treatment are: (1) anatomic rest; (2) muscle reeducation as soon as the inflammatory process has subsided, which may be at the end of the first week; (3) maintenance of warmth of the limbs, massage and avoidance of fatigue and strain.

[ED. NOTE.—The author's principles of treatment are similar to those entertained by the closest observers of our recent epidemics, except that the consensus of qualified opinion seems to suggest a longer period of rest than he imposes. Rarely does it seem wise to begin any muscle training before the second month.]

Lovett [79] points out the importance of recognizing weakness of the gluteus medius and maximus as a controlling factor in many limps following poliomyelitis. Paralysis of the hip flexors and the abdominal muscles also causes limp. The gait should be most carefully analyzed before bone or tendon operations are performed below the hip, with the expectation of getting rid of the limp. Lovett deprecates the performance of serious structural operations on the foot when the chief

77. Saethre: Norsk Mag. f. Lægevidensk. **82**:122 (Feb.) 1921.
78. Mackay: Brit. M. J. **2**:513 (Oct. 2) 1920.
79. Lovett: Surg., Gynec. & Obst. **32**:20 (Jan.) 1921.

cause of the limp lies in the muscles of the hip and abdomen, without a clear statement to parents that, necessary as these operations may be, the limp will not be materially affected by them.

Astragalectomy.—Sever [80] has made an end-result study of 200 cases in which astragalectomy has been performed. The study leads to but one conclusion: that the operation is not a panacea for all cases of calcaneus and "flatfoot," and that it often fails to relieve the limp when the weakness of the hip or of the abdominal muscles coexists. As a result of this analysis, Sever feels that the operation is contraindicated in the case of any foot showing lateral instability as the result of the paralysis of one single muscle group. It is as successful an operation as any for feet which are flail, or in those which have only one muscle group left. If the long flexors are active, varus is liable to develop later, and a bad weight-bearing position results. It is not an operation to be lightly advised or invariably performed for various foot deformities, but should be reserved for older children and selected cases.

Whitman [81] comments on Sever's article, maintaining quite properly that the operation cannot be fairly criticized when judgment is based on a group of cases in which it has been performed to remedy several types of paralysis. He believes that the operation should be uniformly successful in cases of calcaneus, and that only lack of attention to the proper mechanics and insufficient attention to the details of position and after-treatment can account for bad results. If a varus deformity develops, owing to the pull of the tibialis posticus or the toe flexors, it may be overcome by a transplantation of these tendons.

Villard and Perrin [82] describe the results of twenty astragalectomies performed to correct a vicious attitude of the foot, chiefly equinovarus. They believe these results surpass those obtainable by tendon or joint operations.

[ED. NOTE.—Both Sever's and Whitman's articles are valuable, the former as demonstrating that many poor results will follow astragalectomy unless the cases are carefully selected, and Whitman's as emphasizing the fact that astragalectomy was never devised to correct any but a special deformity, and that meticulous attention to mechanical details and after-treatment must be given. If these conditions are met, we believe, as Whitman does, that the results will be all that is desired. However, the very excellence of the results in properly selected cases, in which Whitman's details have been carried out, has led many good

80. Sever, J. W.: Removal of Astralagus in Paralytic Feet, J. A. M. A. **75**:1200 (Oct. 30) 1920.

81. Whitman: J. Orthop. Surg. **3**:19 (Jan.) 1921.

82. Villard and Perrin: Lyon chir. **17**:36 (Jan.-Feb.) 1920.

surgeons to extend the application of this procedure, and their bad results are tending to cast discredit on an operation which in these cases should never have been performed.]

Pes Cavus.—Mayer [83] has made a careful dissection of a specimen of pes cavus and believes that full correction of the deformity is only possible when the deep articular ligaments of the sole have been divided, especially the long plantar ligament. Simple fasciotomies are entirely insufficient.

Pes Equinus.—Gretsel [84] points out that simple tenotomy is often insufficient to correct severe cases of equinus and reports perfect results from the following operative procedure: An incision is made in the midcalf, starting at the level of the head of the fibula, and running distally to the point at which the Achillis tendon becomes distinct from the muscle. The gastrocnemius is divided at this point, retracted and reflected upward. The soleus is now separated from its origin on the fibula and tibia, with careful preservation of the nerves and vessels perforating the upper end of the muscle. The foot is now brought into dorsal flexion, and the gastrocnemius is turned downward again and retained by a few sutures.

Causes of Success and Failure in Tendon Transplantation.—Dunn,[85] in an extremely helpful article, emphasizes the fact that success in tendon transplantation depends on the possibility of reeducation of the transplanted muscle to perform its new function. When a single tendon has been taken to replace one of its own group, success has been the rule. Groups of muscles, not a single muscle of a group, may be used to replace muscles of a different action, but when a single tendon has been taken from a group to replace one not normally in action with it, failure has often resulted. Dunn finds that the most consistent failure has resulted from transplantation of a single peroneal tendon to replace the tibialis anticus. Both the longus and brevis should be taken when this type of transference is undertaken.

Spastic Paralysis; Stoffel's Operation (Neurectomy).—Gill [86] describes the purpose of the preliminary treatment and after-treatment, the indications, and the technic of the so-called Stoffel's operation or neurectomy for the relief of spastic paralysis. He has operated on thirty-two patients, some of whom have been observed for six years. The youngest was 3 years of age and the oldest 27. No ill results followed the operation in any case. The operations on the popliteal nerve for relief of contracture of the Achilles tendon have been most uniformly successful. The operations on the obturator nerve have,

83. Mayer: Ztschr. f. orth. Chir. **38**:80, 1919.
84. Gretsel: München. med. Wchnschr. **67**:1412 (Dec. 3) 1920.
85 Dunn: J. Orthop. Surg. **2**:554 (Oct.) 1920.
86. Gill: J. Orthop. Surg. **3**:52 (Feb.) 1921.

in all instances, corrected the adductor spasticity. The results in the hand, especially on the median nerve, have been more uncertain. Contractures may be overcome by this operation, but the amount of functional improvement depends on the nature and severity of the disease, on the mentality of the patient, and the careful, persistent after-treatment.

Tietze and Foerster [87] report that resection of the obturator nerve has given excellent results in four cases of adductor spasm. They believe it advisable to resect the nerve within the pelvis at the point at which it enters the obturator foramen, rather than in the adductor triangle, where bleeding from the venous plexus, deep in that region, may be hard to control. In the pelvis it is still undivided; in the abductor region there are two branches.

Léri and Gay [88] call attention to a form of spastic paraplegia prone to follow abortive epidemic encephalitis.

De Stefano [89] reports five cases of spastic paraplegia occurring in two families with syphilitic taint. He urges intraspinal, antisyphilitic treatment before degenerative changes have occurred.

Paralysis of the Iliopsoas.—Eberstadt [90] has found Ludloff's symptom of great value in diagnosing cases of isolated paralysis of the iliopsoas. When the patient is sitting upright on a table with the knees extended he is unable to raise his leg unless the iliopsoas is active. In that position the other flexors of the hip, the sartorius, the rectus, and the tensor fascia femoris, are thrown out of action.

Tendon Transplantation in Radial Paralysis.—Achard and Jarkowski [91] report excellent results from extensive transplantations after the manner of Jones [92] and Starr,[93] in cases in which irreparable damage and failure of repair of the radial nerve have occurred. They admit that these results are inferior to those in which radial continuity and subsequent repair have occurred, and council against the performance of these transplantations until every hope of this regeneration must be abandoned. They state that, when conditions are favorable, the central stump grows toward the periphery at the rate of 1 or 2 mm. a day. The integrity of the median and ulnar nerves must be complete before any transplantation operation is attempted. They advocate shortening the extensor tendons in the forearm, thereby avoiding an incision on the back of the hand, and lessening the danger of adhesions.

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87. Tietze and Foerster: Zentralbl. f. Chir., 1920, p. 400.
88. Léri and Gay: Bull. et mém. Soc. méd. d. hôp. de Paris **44**:876 (June 18) 1920.
89. De Stefano: Pediatria, Oct. 1, 1920.
90. Eberstadt: München. med. Wchnschr., No. 30, 1920, p. 1020.
91. Achard and Jarkowski: Progrès. méd. **35**:387 (Sept. 4) 1920.
92. Jones: Brit. M. J., March 28, 1908.
93. Starr: To be published.

TENDON, BONE AND JOINT SURGERY

Heterogenous Tendon Grafts.—Durand [94] calls attention to the wide field for tendon implants which Nageote and Sencert (and he should have said Gallie) have opened up. The two former investigators believe that tendons taken from the embryo in utero of a cow are peculiarly adapted for grafting work, and that the fibrous portion of the tendon is all that is needed for the implant. The cells of the living organism into which the tendon is implanted grow into this fibrous framework with great ease. In preparing the implant, it is slightly stretched on a glass rod by means of threads passed through each end, and placed in 90 per cent. alcohol and sealed for ten days. The alcohol is now changed to 60 per cent. and the tube sealed anew, ready for use. They have used tendons thus preserved for seventeen months and have found them entirely satisfactory. Before the tendon is used, the alcohol is rinsed out with tepid artificial serum; the implant is sutured in place with fine catgut, and the limb is immobilized in plaster to relax the tendon. In Sencert's thirty-five grafts, the implanted tendon was never cast off.

Jaliper [95] also reports no failure in five cases in which implanted tendons from calves and dogs, removed from six to fifty days previously, had been employed for repair of hand tendons. The function is fairly good or very good in all.

Tenotomy.—Vulpius [96] believes that simple tenotomy is not such a harmless operation as the textbooks would lead one to believe. Examination of patients who have had section of the Achilles tendon performed frequently reveals improper length of the tendon, adhesions to surrounding tissue, etc. The Z-shaped, step-like tenotomy of Bayer injures also the gliding tissue, although overcoming some of the other dangers of simple tenotomy. Vulpius' method of procedure, which has given him great satisfaction in section of the Achilles tendon, is as follows: An incision is made along the border of the gastrocnemius, where it becomes tendinous, and the muscle and tendon are exposed. Incisions are made in the flat tendon with a V-shaped apex downward. A bit of the tendon running into the muscle belly is divided, the foot dorsally flexed to the desired angle, and the fascia and skin closed, without any sutures being taken in the muscle or tendon.

Dupuytren's Contracture.—Momburg [97] is in favor of conservative treatment of Dupuytren's contracture, in contrast to the more recent literature on the subject. In early cases, he has found persistent stretchings carried out by the patient to be efficient. In the more

94. Durand: Lyon méd. **129**:852 (Oct. 25) 1920.
95. Jaliper: Lyon chir., January and February, 1921.
96. Vulpius: München. med. Wchnschr., No. 15, 1919, p. 416.
97. Momburg: Deutsch. med. Wchnschr. **46**:602 (May 27) 1920.

advanced cases, he makes subcutaneous incisions of the prominent bands and has never been obliged to resort to the radical procedures which he considers dangerous to future function.

Repair of Lateral Ligaments of the Knee.—Edwards,[98] impressed by the success of two war cases of instability of the knee, describes the operation which he worked out on the cadaver. He believes that the function of the collateral ligaments is more important than that of the crucials, and that if these lateral ligaments are strong and tight, satisfactory function results. The operations are performed with the knee slightly flexed. In repairing the external lateral ligament, a lateral vertical incision is made and a rectangular flap from the fascia lata about 3 inches long is turned down. A similar flap of the deep structures, chiefly the tendon of the biceps left attached below, is defined, and pulling the fascial flap down and the tendon flap up, they are implanted into grooves in the condyle of the femur and the head of the fibula, fixing them with staples. For the internal or mesial ligament, a vertical incision is made over the mesial condyle, exposing the sartorius and the tendons of the gracilis and semitendinosus. These are separated from the condyles, pulled forward, and divided at the level of the condyle. The distal portions of the tendons are stitched together, and they are together implanted into a groove in the condyle of the femur and fixed under tension by a staple. The deep structures are sutured over them. The proximal portions of the tendons are stitched to the sartorius in order to preserve the function of the semitendinosus and gracilis. The sartorius is drawn well back to preserve the normal line of pull, and the wound is closed.

De Frenelle [99] reports similar procedures, using heterogenous tendon grafts in addition, if they are required in case of actual luxation.

Transplantation of Bone.—Leriche and Policard,[100] after extensive study of the fate of implanted bone, assert that the term "graft" cannot be applied to transplantation, because the implant inevitably loses its individual vitality. All bone transplants die. A fresh autotransplant provides the best material for regeneration when embedded in receptive tissue. Bone does not form well when there are great numbers of polymorphonuclear cells, in other words, in the presence of pus. They believe that the bone marrow of the implant plays no appreciable part in the regeneration. If the surrounding tissues are fibrous and avascular, there will be no reconstruction of bone. The reconstruction occurs by new intrahaversian formation, and, in the case of bone periosteum transplants, takes from twelve to fifteen months. Function of the limb, as soon as absorption has begun, is the best stimulus to

98. Edwards: Brit. J. Surg. **8**:226 (Jan.) 1921.
99. De Frenelle: J. de chir. **16**:399, 1920.
100. Leriche and Policard: Lyon chir. **17**:409 (July-Aug.) 1920.

regeneration. Their best results in treatment of pseudarthrosis of the tibia were in two cases in which weight bearing was allowed a month after the implantation. They consider it a biologic error to implant a hard solid bone in the hope of securing a stronger support. Slightly compact bone with the periosteum well contacted, is best fitted for the rôle it must play, the aim being to assure the absorption of the transplant in the shortest possible time. Implantation into spongy bone is much more likely to succeed than implantation into compact bone. Their conclusions are based on the ultimate outcome in nineteen cases. Bone periostum transplants can be successfully employed in gaps of from 12 to 15 cm., but in gaps of this length they believe it wiser to make use of a collection of bone-periosteum slices rather than a single block, except when the temporary support of a more solid graft is needed, as with the humerus or the femur. They look favorably on the implantation of previously prepared dead spongy bone, and believe the failures of the method have resulted from the employment of too hard and compact dead implants.

Cotte [101] also urges the bone-periosteum grafts as a routine, reserving the Albee transplant for very extensive defects. Like Leriche in the pervious article, he is impressed by the abhorrence which bone implants have for metal plates and wire.

Eloesser,[102] in an extremely judicial and careful article, discusses the employment of autotransplants of the ribs, basing his report on a series of twenty-two cases. He adopted the rib because it possessed certain theoretical advantages. It has a thin cortex and a large proportion of spongy tissue, lending itself to rapid absorption. Gallie and Robertson have shown that the process of absorption takes place more rapidly than the process of replacement. Therefore, the denser cortical tibial implant would be more suitable in cases in which stress and strain exist. No special instruments are required for removal of the rib. It can be readily cut and bent, and pierced with a needle. The supply is unlimited and the patient is not exposed to any risk of subsequent fracture. Eloesser's technic is to remove the rib with its periosteum, split it, and wrap it about the freshened bone ends, retaining it with kangaroo tendon passed through drill holes. His conclusions are: 1. Rib implantation is a feasible procedure. 2. The viability of the rib implants is great, even in the presence of suppuration, and they are more likely to survive in the presence of infection than more massive implants. 3. They are more rapidly absorbed. 4. They hypertrophy slowly. 5. They are prone to refracture (seven out of twenty-two cases). 6. They are not so useful as tibial implants

101. Cotte: J. Médécine **2**:15 (Oct.) 1920.

102. Eloesser, Leo: Rib Grafting Operations for Repair of Bone Defects and Their End-Results, Arch. Surg. **1**:428 (Nov.) 1920.

for the repair of large defects or when the implant is to be put under strain. 7. They are particularly useful when no great demand is to be made on the strength of the implant, as in repair of the bones of the skull and in facial plastics. 8. In twenty-two cases there were three failures, six partial successes, and thirteen successes.

Hamilton,[103] in cases of pseudarthrosis, implants his grafts under traction, cutting the proximal and distal ends on a bevel. The receptive channel is also prepared with overhanging edges and the implant sprung into place while traction is exerted on the limb. Greater firmness and better contact are the advantages of the method.

Magnuson [104] employs bone keys of ivory in fixing bone ends on account of the great strength of the ivory, and its slow but sure absorption and nonirritating qualities. After preparing with twin parallel saws a key way passing down into the medulla, a hole is drilled at each end. The ivory key is driven firmly into the key way and held in place by ivory screws passing at right angles to the key.

Shoulder Defects.—Albee [105] calls attention to the helpless condition in which patients who have suffered defects of the upper portion of the humerus are left, and the comparative frequency with which these cases are met. The surgeon must decide in the individual case whether an attempt should be made to preserve and perhaps increase motion between the scapula and the humerus, or whether an arthrodesis would result in more useful function. If motion is to be sought for, this motion must be controlled by muscles, else it is of less advantage than complete ankylosis between the scapula and the humerus, and the well controlled motion of the scapula is made available to move the humerus as well. In attempting to provide a movable scapulohumeral joint, Albee employs the head and a small portion of the shaft of the fibula, driving it for the length of an inch into a drill hole in the humerus, which may need to be split as well, if the circumference of the fibula is considerable. Albee emphasizes the importance of stitching the rotator and elevator muscles of the shoulder to the humerus and fibula implant in as nearly normal position as possible. If ankylosis is the end sought, he employs two fairly large and strong grafts in a sort of truss work, one from the humerus into the acromion and from the glenoid to the humeroacromial graft. These grafts are firmly and accurately fitted into their host bones, while the arm is held in the exact position the operator desires to maintain. The most favorable position is considered to be with the humerus elevated anteriorly and rotated so that the hand is brought in front of the face.

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103. Hamilton: Texas State J. Med. **16**:210 (Sept.) 1920.
104. Magnuson: Northwest Med. **19**:299 (Dec.) 1920.
105. Albee: Surg., Gynec. & Obst. **32**:1 (Jan.) 1921.

Nonunion of the Humerus.—Henderson [106] reviews forty-one cases of pseudarthrosis of the humerus, finding the common cause to be imperfect immobilization in ambulatory splints. He advocates the use of the massive graft on the basis of his comparative results and because more endosteum to endosteum contact is thereby obtained. He thinks the medullary graft unsound in principle. His results show 85 per cent. of good results from the massive graft (fibula?), 70 per cent. from the inlay, and 50 per cent. from the intramedullary peg.

Ankylosing Operations on the Spine.—Polya [107] has been employing with satisfaction a modification of Albee's method of inserting a spinal graft. The erectores spinae on one side are dissected from the spinous processes which are divided as near their bases as possible. Still attached to the muscles on the other side, they are turned back, and the bony bed smoothed for the reception of the graft. The spinous process muscle flap is then adjusted over the graft, and the fascia and skin sutured. The technic is simple, and the graft, well contacted, is brought nearer the laminae thereby.

Forbes [108] describes his method of ankylosing portions of the spine, chiefly in selected cases of scoliosis, not claiming that it represents the perfection of Hibbs' operative technic, but maintaining that it thoroughly accomplishes its purpose. In the dorsal region, the spinous processes and laminae are exposed and so gouged by a concave chisel that their cortical layers are separated from the medulla beneath. A series of bone chips and fragments of periosteum are pried up along the superior and inferior borders of the laminae and made to interdigitate with one another. In the lumbar region, the spinous processes are split vertically into several thin slices, and bent upward and downward and to the sides, meeting each other and osteoperiosteal chips from the laminae. Forbes aims to create a veritable forest of bone chips. The plaster jacket applied in the best possible position obtainable is worn for six months.

New [109] reports fifteen operations in which he has employed boiled beef bone as the inlay for ankylosing a portion of the spine in cases of Pott's disease. He says the results are satisfactory as far as he knows them. The operative technic is that of Albee. The graft was retained in all but one case. This patient got out of bed the second day after the operation and the wound was split open, with subsequent infection.

[Ed. Note.—After a rather extensive experience with this method (thirty cases) and a careful study of end-results at the orthopedic clinic of the Massachusetts General Hospital, Brown considers the

106. Henderson: Southern M. J. **14**:148 (Feb.) 1921.
107. Polya: Zentralbl. f. Chir. **48**:884 (June 25) 1921.
108. Forbes: J. Orthop. Surg. **2**:509 (Sept.) 1920.
109. New: Nat. M. J. China, March, 1920.

value of this operation as still sub judice. The graft is always well tolerated; it splints the spine quite firmly at first, if it is accurately placed and retained, and it absorbs very slowly, but the living bone of the host seems to replace it slowly also, one may almost say rather reluctantly. It has been recovered a year and a half after insertion, almost unchanged, lying without irritation, but without fusion to its bed. Its use has many advantages, but our impression is that it cannot, in a certain proportion of cases, be counted on to fix, firmly and permanently, the vertebrae into which it is implanted, and that some form of fusion operation is necessary in addition. It may well prove a useful, nonirritating temporary splint favoring early fusion, and affording immediate fixation. Experiments on animals now in progress will confirm or refute this impression.]

Ankylosis of Sacroiliac Joint.—Hertz [110] describes a method of immobilizing the sacroiliac joint by driving a bone peg into it across its articular surfaces. He has employed the method in three cases of suppurating and in one case of nonsuppurating sacrocoxalgia. The implant healed without irritation.

[Ed. Note.—It is quite possible on the cadaver, as Hertz says, thus to immobilize the sacroiliac joint by driving a bone peg through it. We doubt the permanence of this immobilizing procedure, however, in the living subject, since the peg traverses an articular space. The procedure of Smith-Petersen seems to us to be better conceived. He gains access to the sacroiliac joint by removing a carefully preserved rectangular plug of bone from the ilium, running at right angles to the joint. He then denudes the joint of cartilage, continues his window into the sacrum, and drives the plug of bone into it, setting it like a nail beneath the surface of the ilium. He has performed this operation twelve times, with almost complete success. A fuller report will be made in a later report of progress.]

Transplantation of Fibula for Large Defects in Tibia.—Brandes [111] reports four cases of transplantation of the fibula into the tibia for large defects. If the defect reaches into the upper tibial epiphysis, he transplants the whole head of the fibula with the shaft, for purposes of growth. The upper end is transplanted first and then the lower, leaving the external malleolus, but bringing it in contact with the transplanted shaft. He counsels leaving the fibula longer than the apparent defect, as it sinks well into the spongy medulla of the tibia.

[Ed. Note.—It will be remembered that Stone, Codman, Campbell, MacAusland, and Wood have all reported similar successes, the fibula hypertrophying quite rapidly, finally attaining almost the size of a normal tibia.]

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110. Hertz: Lyon chir. **17**:199 (March-April) 1920.
111. Brandes: Deutsch. Ztschr. f. Chir. **155**:312, 1920.

Arthroplasty.—Payr [112] reports a case of bony ankylosis of the hip, associated with partial fibrous ankylosis of the knee, ankle, and elbow. Arthroplastic operations were performed on all the joints with success.

Putti [113] epitomizes his experience with arthroplastic operations on the knee and thus emphasizes what he has grown to consider the essentials of the procedure. 1. Complete removal of all intra-articular structures is necessary. 2. Operation too soon after acute inflammation spells failure. 3. In joints ankylosed as a result of Neisser infection the postoperative treatment should be very gentle. A longer time is required in these cases for good results. 4. The knee on which the operation has been performed usually remains larger than the normal knee.

Osteochondritis Dissecans.—Rieger [114] agrees with Koenig and Axhausen in regard to the pathogenesis of osteochondritis dissecans, believing that an actual dissecting process takes place, and combating the contention that an actual single or even repeated trauma mechanically expels the bit of bone. The opinion which Rieger upholds is that of an actual fat embolism in the tiny end arteries of the lower end of the humerus and femur and the ends of other long bones. Fat embolism of these arteries has been observed at necropsy. In adults, these end arteries run in fat cells, in growing children, in connective tissue; and Rieger believes this conception of the pathogenesis of the condition explains the absence of the condition in children.

Kappis [115] believes, on the other hand, that the affection is always of traumatic origin, although he admits that apparently the blow is not always sufficient to cause immediate disruption of the fragment into the joint. He thinks that loose bodies in joints usually are derived from marginal proliferations or pieces of articular cartilage, and are of an arthritic nature. The bony tissue becomes necrotic, the marrow remains intact, and the hyaline cartilage is transformed into fibrous cartilage, continuing to grow without perichondrium in a way similar to that of experimental growth of tissue in nutritive fluid.

Axhausen [116] believes also that in the so-called osteochondritis dissecans, trauma is the important element; but he thinks the separation takes place slowly. The particular trauma which causes it is generally a blow on the condyles of the femur, with the knee flexed. In the cases in which operation had been performed soon after a trauma, and the separated bone found, the microscopic findings suggest a process antedating by a long time the last trauma. In his own case, the process was

112. Payr: Arch. f. klin. Chir. **114**:878, 1920.
113. Putti: J. Orthop. Surg. **2**:530 (Sept.) 1920.
114. Rieger: München. med. Wchnschr., June 15, 1919.
115. Kappis: Deutsch. Ztschr. f. Chir. **157**:214, 1920.
116. Axhausen: Deutsch. med. Wchnschr. **46**:825 (July 22) 1920.

almost entirely one of resorption with very active connective tissue and giant cells eroding the surface of the necrotic bone. In this case, the body was found free immediately after a trauma, and the casual observer would have considered it the immediate result of the last injury. Axhausen ventures the hypothesis that the dissecting process may be due to an embolus, perhaps a tuberculous one. A trauma only completes the separation.

Derangements of the Semilunar Cartilages. — Strahlmann and White,[117] reviewing the histories and operative findings in about sixty cases of derangement of the similunar cartilages of the knee, consider the most reliable diagnostic signs to be (1) a traumatic history, with recurring symptoms of disability; (2) definite localized pain or tenderness; (3) locking; (4) sense of derangement or feeling of insecurity; (5) swelling. They believe that the simple untorn hypermobile cartilage is an entity more common than fracture, and that it requires surgical intervention. Excision should be as complete as possible under perfect asepsis. The prognosis should be more or less guarded in cases of long standing, especially in those in which at operation the cartilage is found lacerated or comminuted. The reason for this is that secondary joint changes have occurred and are not always relieved by the removal of the original cause.

Jacobs and Worms [118] think that if a dislocated cartilage can be immediately reduced, and the limb immobilized and massaged while the patient practices voluntary exercises of the quadriceps, most patients fully recover. They have analyzed the results in 200 operative cases and believe the operation of meniscectomy entirely satisfactory in actual crushes or fractures of the cartilage or in the presence of recurring symptoms. Resection of small portions of the cartilage often fails to relieve.

Terracol and Colanéri [119] have injected air directly into the knee joint, following injuries from which damage to the semilunar cartilages was suspected. They feel that they are able to detect fractures and tearings by this method in the roentgenograms, confirming their clinical findings.

[Ed. Note.—We feel that lesions of the semilunar cartilages do not usually present great diagnostic difficulties. At least, the advisability or nonadvisability of operative exploration is usually easy to determine. We should doubt the wisdom of injecting air into a recently traumatized joint for the sake of possibly obtaining preoperative information, which exploration will more surely reveal.]

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117. Strahlmann, L., and White, J. W.: Derangement of Semilunar Cartilage, J. A. M. A. **76**:561 (Feb. 26) 1921.

118. Jacob and Worms: Rev. de chir. **39**:898, 1920.

119. Terracol and Colanéri: Presse méd. **29**:113 (Feb. 9) 1921.

PERIPHERAL NERVE INJURIES

Reviews of Peripheral Nerve Injuries.—Stuckey [120] emphasizes the importance of reconstructing the original injury in an effort to evaluate the benefit likely to be derived from operations looking to the return of peripheral nerve function, and also for the purpose of diagnosing the existing type of injury to the nerve. In charting the sensory findings, it is not enough to use the terms epicritic and protopathic, but the type of stimulus used and the response thereto should be given. In examining for light touch, longitudinal, not transverse, strokings should be made, else confusion is probable. Stuckey states that the dorsal surface of the terminal phalanx of the thumb is the only area exclusively supplied by the radial nerve. In examination for return of voluntary motion, he pleads for estimation by sight and touch of individual muscle action as opposed to group action and motion of joints. The risk of being deceived by trick movements is thus materially lessened. The author recommends early and extensive nerve exploration in the majority of cases, but advocates great conservatism in suture, etc., until all facts as to late results are weighed.

In studying the true end-results of operations on the peripheral nerves, Thorburn [121] urges the recorder to group under headings what he means by restoration, that is, anatomic, physiologic, or economic restoration. The completeness of recovery of a divided nerve, Thorburn believes, is proportionate to the accuracy with which the individual fibers of the central or motor cable are approximated to these same fibers in the peripheral segment, and when there has been hopeless entanglement of motor, sensory and trophic filaments, there must be corresponding physiologic failure and confusion. When operative technic is accurate, good results may apparently be expected in about 50 per cent. of the cases (from one-third to two-thirds), but a perfect neurologic result is almost never attained. The chances of recovery are better when the small muscles do not have to be reenergized, and when the given nerve is of an almost purely motor or sensory type. The nearer the section is to the spinal cord, the better the prognosis. In the upper limb, the radial nerve is an easy first in its power of recovery, the median a bad second, and the ulnar a very poor third; but it is interesting to note that the absence of ulnar power sometimes cripples the hand very little. A surgeon who obtains 50 per cent. of really good economic recoveries after secondary nerve suture must be doing, according to our present standards, first-class work.

Sargent [122] points out the many factors which, in war wounds, militate against complete recovery from nerve suture. The most important

120. Stuckey: New York State J. M., September, 1920.
121. Thorburn: Brit. M. J. **2**:462 (Sept. 25) 1920.
122. Sargent: Brit. M. J. **2**:464 (Sept. 25) 1920.

of these are the concomitant vessel, muscle, tendon, and bone injuries, complicated by sepsis, which in many cases have resulted in an intraneural fibrosis, ascending and descending, and opposing the advance of the down-growing axons. End-results are of value only as they record the effect of end-to-end suture, carried out by skilful operators. Apparently the results are as good after secondary suture as after primary suture, other things being equal. Sargent, in conclusion, discusses the relief of causalgia by alcohol injections, section and resuture, and division of the sensory roots.

Forrester-Brown's [123] study of end-results of operations on peripheral nerves is based on 643 cases of nerve injuries observed at the Edinburgh War Hospital. The basis on which these were studied was (a) capacity for work, and (b) degree of recovery after neurolysis and suture. In 237 cases of suture, 12 per cent. of the patients are at ordinary work, 35 per cent. at light work, and 15 per cent. at heavy work. In 187 cases of neurolysis, 80 per cent. of the patients are at ordinary work. In eighty-one cases of tendon transplantations, 23 per cent. of the patients are at ordinary work, and 36 per cent. at light work. As to recovery after suture, of 158 patients, 28 per cent. showed complete motor return, 19 per cent. complete sensory return and 21 per cent. complete trophic return. Fifty per cent. showed incomplete return of all these functions. Total complete recovery of all functions was found in only five of the median cases, and in eight of the musculospiral. Three nerve grafts were explored because of lack of improvement. The graft could with difficulty be recognized, embedded in scar tissue, and of small caliber. There was no reaction to the faradic current. In 117 cases of neurolysis, 42 per cent. of the patients had full motor and functional return and 19 per cent. full sensory return. The early functional improvement after tendon transplantation was impressive and satisfactory. As a result of this study, the author believes that Tinel's sign should be discredited as an index of progressive recovery, and that the degree of recoverability is not dependent on the length of time which has elapsed between injury and suture.

Corbett [124] makes the interesting statement that in 100 cases of nerve injury from war wounds, 60 per cent. of the patients recovered without operation. He has accepted as the indication for exploratory operation three months of lack of improvement under proper conservative treatment, splinting, etc. In performing the operation of neurolysis, Corbett believes that it is very important to make fine longitudinal incisions into the nerve itself as well as to free it from surrounding

123. Forrester-Brown: Brit. M. J. **2**:467 (Sept. 25) 1920.
124. Corbett: Minnesota Med. **3**:422 (Sept.) 1920.

tissue. An internal keloid was demonstrated in several of the cases. Fat and muscle beds are considered to be most suitable as locations for the restored nerve.

Pollock [125] had an opportunity to make a bed to bed observation of cases of nerve injury in the hospitals of the American Expeditionary Forces. The striking feature was the very great percentage of improvement without operation. He estimates that there were 10,000 cases of nerve injury among our forces, and of that number only about 3,000 came to be classified as such in the reports of the Surgeon-General's office, indicating that approximately 66 per cent. of the patients had recovered sufficiently before they returned to America to be no longer classified as nerve cases. He estimates also that not more than 14 per cent. came to operation. These figures are not far from Corbett's estimate in the previous review. Like Forrester-Brown, he believes that Tinel's sign of tingling on distal tapping is not to be depended on in prognosis of recovery after suture. The duration of the paralysis without signs of beginning recovery is the best indicator by which to correlate the questions of physiologic and anatomic interruption.

Harris [126] reports the success of an operation to relieve a median anesthesia of the fingers. The lesion was distal to the muscular branches. The anesthesia was very troublesome, preventing the man from buttoning his coat, etc., and causing numerous burns. An anastomosis of the radial nerve into the distal portion of the median was performed. Sensation returned slowly, but the return was finally complete.

Barling [127] reports a case of a bad wound of the root of the neck with extensive injury to the brachial plexus. This was followed by almost intolerable pain in the arm for more than a year, apparently due to involvement of the plexus in the scar. For relief of the condition, complete intrathecal division of the fifth, sixth, seventh, and eighth cervical, and the first and second dorsal spinal roots was performed. The relief was immediate and permanent.

OSTEOMYELITIS

Bone Necrosis and Suppuration.—Ritter [128] takes issue with the textbooks of pathology and surgery, which nearly all teach that suppuration causes bone necrosis, by maintaining that it is the necrosis which causes the suppuration. To support his contention of this sequence, he calls attention to the fact that, even in the early cases of osteomyelitis, necrosis cannot be prevented by drainage operations. It is always present at operation and rarely increases. Suppuration of the

125. Pollock: Ohio State M. J. **16**:120 (Oct.) 1920.
126. Harris: J. Orthop. Surg. **2**:519 (Sept.) 1920.
127. Barling: Brit. J. Surg. **8**:142 (July) 1920.
128. Ritter: Arch. f. klin. Chir. **114**:1013, 1920.

bone marrow need not necessarily exist. There are certain spots of predilection for osteomyelitis, which suggest that the necrosis is determined by the arrangement of the arteries. In the growing child, as long as the epiphysis persists, the diaphyseal and epiphyseal arterial systems are almost entirely independent of one another, as the researches of Lexer and his pupils have shown. Although there is some collateral circulation, these arteries are to be considered functionally as end arteries. In the growing person, the amount of necrosis is determined by the arterial system, into which the bacterial embolus enters. In the adult, the epiphyseal and diaphyseal arteries communicate with each other much more freely, and the pictures of osteomyelitis in children are very different from those in adults. If Ritter's facts are true and if his reasoning is sound, the article has an important bearing on treatment. It would seem not only to be unnecessary, in many cases, to open the medullary cavity, but also to be dangerous to do so, unless necrosis within the cavity could be proved. He claims to have cured many patients suffering from early osteomyelitis by single or repeated simple drainage operations, sequestration never occurring. Bier is quoted as having reached similar conclusions.

[ED. NOTE.—It will be remembered that in the fifteenth Report of Progress we commented on a collection of articles advocating conservative rather than radical bone operations in early acute osteomyelitis on the basis of results obtained by these conservative methods. One of the editors has performed an operation in a case of early osteomyelitis attacking the tibia of a child. The abscess was evacuated by a free incision. The tibia, which was necrotic to a large extent, was not touched. The wound was closed without drainage as an experiment. Healing by first intention occurred, and no sequestration. Another of the editors has observed a similar case, also occurring in the tibia, following measles. In this case, a small opening into the bone near the upper end of the tibia had been made before the patient was seen. The roentgenogram revealed an involvement of almost the entire shaft, which appeared necrotic. There was no temperature or pain and very little discharge. The small opening in no way provided ample drainage for the shaft, but the case went on to complete reformation of the tibia and complete healing, without further operation, except the removal of a small sequestrum which was lying loose in the head of the tibia at the point at which the first opening into it had been made. We are not prepared to advocate such extreme conservatism as a routine, but the cases seem suggestive in the light of Ritter's article.]

Clinical Considerations of Osteomyelitis.—Ochsner and Crile[129] review the gross and microscopic anatomy, physiology, and pathology of bone as necessary to an understanding of the osteomyelitic process.

129. Ochsner and Crile: Surg., Gynec. & Obst. **31**:263 (Sept.) 1920.

The conditions of blood circulation in bone make easy the formation of abscesses, because the nature of the tissue allows for no swelling, and surrounding inflammation and infarction result. These authors maintain that in acute cases the medulla should invariably be freely opened, and hot packs and dry heat should be subsequently applied. In well advanced severe cases, however, they believe surgical intervention should be confined to opening the periosteum throughout the whole area of infection. The use of hot packs and dry heat should follow. In chronic cases complete surgical obliteration of the cavity and removal of all sequestrums is the procedure to be followed, preceded by a search for all possible sources of focal infection elsewhere in the body.

Kidner [130] and Ryerson [131] hold much the same views as to treatment as Ochsner and Crile. Ryerson has been pleased with the results of secondary closures in chronic cases in which, after "saucerization" of the cavity and Dakin-Carrel sterilization, a muscle and fat flap has been turned in.

Heinemann [132] describes his successful closure of bone cavities by the following technic: During the week preceding the operation the bone cavity is cleaned and thoroughly rubbed with tincture of iodin. The sinus is exposed widely through normal tissue if possible. The bone cavity is freely exposed so as to be accessible in all its extent. This cavity is next curetted and wiped out with tincture of iodin. The next step is to cover the soft tissue with wet compresses while the cavity is thoroughly dried by a hot air blower. The hot air vaporizes the iodin, enhancing its antiseptic qualities and thoroughly removing it from the cavity. A flap of fat tissue cut out of a suitable part of the patient previous to the handling of the cavity is now made to fill the sterilized cavity and the soft tissues closed in two layers over the fat. Three cavities of the humerus have been thus closed successfully. He attributes his success, as did Mosetig-Moorhoff, to the peculiarly careful sterilization of the bone cavity before, and at the time of, operation.

AMPUTATIONS

Corner [133] calls attention to the great simplification of amputation operations which the unfortunately large number of war cases has brought about. Discarding the elaborate and varied textbook amputations, he believes that in the lower limb there remain to be advised and practiced only (1) the Syme operation; (2) lower leg amputation 4 or more inches (10 cm.) above the ankle joint with ample skin flaps; (3) amputation of the thigh with long anterior and short

130. Kidner: J. Orthop.Surg. **2**:470 (Aug.) 1920.
131. Ryerson and Beeson: J. Orthop. Surg. **2**:499 (Sept.) 1920.
132. Heinemann: Arch. f. klin. Chir. **113**:811, 1920.
133. Corner: Lancet **1**:114 (Jan. 15) 1921.

posterior skin flaps, and (4) a new amputation through the neck of the femur. In the arm, (1) amputation of the forearm with equal skin flaps; (2) circular amputation of the upper arm, and (3) Spence's amputation at the shoulder are practicable.

Cathcart [134] thinks well of the classical Chopart's amputation in the foot and of amputations through the knee joint.

[ED. NOTE.—Chopart's amputation was found generally unsatisfactory by most surgeons during the war, and we see no immediate reason for passing over this experience, but amputations through the knee joint, which have also been somewhat discredited, may have become so because of lack of experience with them. It is generally agreed that in very high lower leg amputations, the best prosthetic result can be obtained by flexing the short stump and bearing weight on the bent knee. Amputation through the knee joint is certainly a less shocking operation than bone section; and if the knee joint is infected, as it frequently is, this method of procedure avoids opening up new channels and provides drainage. It is possible that this mode of amputation should have a definite place.]

Borchgrevink [135] gives testimony to the value of excision of the fibula in all amputations below the knee. The method was employed during the war in the American Army, in all cases in which there were short stumps, and unquestionably it provides a better weight-bearing surface for an artificial limb, by making available the under surface of the external tuberosity of the tibia.

Rockey [136] reports a case of cinematization of the biceps tendon of the arm by the tunnel motor method, in which an excursion of 2 inches (5 cm.) and an ability to lift 75 pounds (34 kg.) was obtained. A satisfactory prosthesis was furnished the elbow, the motion of which was controlled by the opposite shoulder, while the fingers at rest kept open with a spring, can be closed by the biceps motor.

Salomon [137] has been employing with satisfaction a modification of Trendelenburg's procedure for persistent pain and paresthesia following amputation. The nerve is exposed in the stump, its end covered with a thin lead pencil hood and frozen for four minutes. It is then allowed to thaw, and the freezing process repeated once. Pain may be severe for a few days, but has been permanently relieved in all but two cases which were of the neuralgic type, suggesting a more centrally located cause.

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134. Cathcart: Edinburgh M. J. **25**:281 (Nov.) 1920.
135. Borchgrevink: Ann. Surg. **71**:697 (June) 1920.
136. Rockey: Northwest Med. **19**:209 (Aug.) 1920.
137. Salomon: Deutsch. med. Wchnschr. **46**:1390 (Dec. 9) 1920.

Todd,[138] in the course of a very good discussion of the pathology of amputation stumps, says that he has employed for six years the alcoholic injection of the nerves, and advocates it most strongly.

NEOPLASMS

Total Excision of Clavicle for Sarcoma.—Coley [139] presents the statistics of eighteen personally observed and eighty-seven collected cases of sarcoma of the clavicle, in which the operation of total excision had been performed. Sixty per cent. of the cases occurred before the age of 30, and there was a history of trauma and rapid growth in the majority. The tumors of the clavicle are as a rule of central origin. Coley pleads for the operation of total excision, stating that the operative mortality in these cases was about 8 per cent., and that the functional disability following and the deformity were slight. Postoperative treatment by radium in combination with Coley's toxins he considers important. Only eight out of the 105 patients whose cases he reports have been apparently cured. The course is very rapid, death ensued in several cases five to six months after the tumor was first discovered.

Cofield,[140] discussing the difficulties in the diagnosis of osteosarcomas, considers the deep boring pain, often severe and unrelieved by fixation, to be the most characteristic symptom. He states that the pain of the periosteal sarcomas is likely to be less severe than that of the medullary type.

Blume [141] reports that 37 per cent. of twenty-eight patients having sarcoma who showed retrogression under roentgenologic treatment have been free from all symptoms for periods varying from eighteen months to five years. Twelve of the twenty-eight patients showed recurrences; seven were unaffected by treatment. When there was recurrence, the seat of the tumor was not involved.

Myxomas.—Bloodgood,[142] reviewing his own cases and those of his colleagues at Johns Hopkins, finds that, in every case of myxoma in which the tumor has been removed "piecemeal," there has been recurrence, and that several patients have died of metastases. He believes, therefore, that myxoma must be classed as a malignant tumor and that the presence of myxomatous tissue in any bone tumor is an indication for active radical treatment. If complete excision is to be attempted, there should always be microscopic examination of the margins of the growth. Amputation may well be the safest procedure.

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138. Todd: Brit. J. Surg. **8**:88 (July) 1920.

139. Coley: Ann. Surg. **72**:231 (Aug.) 1920.

140. Cofield, R. B.: Some Difficulties in Diagnosis of Osteosarcoma. J. A. M. A. **75**:1264 (Nov. 6) 1920.

141. Blume: Ugesk. f. Læger **82**:943 (July 22) 1920.

142. Bloodgood: Ann. Surg. **72**:712 (Dec.) 1920.

Exploration for diagnosis he considers justifiable, with immediate examination by frozen section. He lays great stress on cauterization of the area from which the diagnostic bit is taken, on account of the danger of operative transplantation of cells. The actual cautery or phenol (carbolic acid) may be employed. It is a rare tumor of periosteal or central origin. Of 270 bone tumors, nine were myxomas and three chondromyxomas. At the present time not a single patient in whose case curetting alone was the operation is well and free from recurrences.

Benign Cysts and Osteitis Fibrosa.—Bloodgood,[143] reviewing 158 cases of central primary bone tumors, in which were included chondromas, myxomas, central giant cell tumors, central sarcomas, and bone cysts (osteitis fibrosa), finds fifty-four cases of bone cysts. The microscopic examination of the fibrous lining, connective tissue lining, and the tissue of the dilated haversian canals, is identical, and points to an inflammatory nature rather than to a neoplasm. It is not quite clear why, in some cases, osteitis fibrosa becomes cystic, but apparently the cystic change is the manifestation of the terminal reaction of which the bone is capable, the next stage going on to endosteal reaction and healing. The microscopic picture is sometimes confusing, the cellular areas which sometimes exist in the fibrous tissue in a case of osteitis fibrosa often being responsible for a diagnosis of fibrosarcoma. The natural termination is to spontaneous healing, fracture or a perforation of the cortex. In the latter case the escaped fluid may cause a soft part and periosteal reaction which may simulate sarcoma. Cartilage cells are sometimes found, but never any myxomatous tissue. A rather tough egg-like lining membrane and an absence of hemorrhage suggest a bone cyst. Myxomatous tissue has the appearance of tapioca stippled with blood. The presence of a distinctly hemorrhagic cyst points to malignancy.

It is often difficult to differentiate bone cysts, in the roentgen-ray picture, from myelomas and central sarcomas. The bone cyst generally attacks the shaft, while the myelomas and central sarcomas are more common in the region of the epiphyses. When healing begins, in the case of a cyst, new endosteal bone formation becomes evident, whereas in myelomas and sarcomas these appearances are not found. The bone cysts occur most often during adolescence, and the spontaneous fractures which may occur are painless. Bone cysts do not recur. The so-called recurrences are usually reported on the basis of unnecessary operations undertaken while the slow process of healing is taking place. This process may occupy a period of two or three years. The femur was the most frequent seat in Bloodgood's thirty-three cases, the tibia next, and the humerus third. As to treatment, the author advises

143. Bloodgood: Southern M. J. **13**:888 (Dec.) 1920.

simple protection to prevent fractures, in patients less than 15 years of age. If fracture occurs, it may lead to healing, and it is treated as an ordinary fracture. In older patients, if the condition is progressive with no roentgenologic evidence of ossification, an exploration should be advised, under most rigid asepsis.

Xanthoma.—Merrill [144] reports a case of xanthoma presenting multiple bone lesions. The findings confirm the previously held opinion that the disease is a systemic condition, in which many of the body tissues may be involved, the more prominent skin lesions being only a surface manifestation, the bones and other tissues frequently sharing in the condition.

Multiple Cartilaginous Exostoses.—Honeij's [145] detailed analysis of sixty-six cases of multiple cartilaginous exostoses has shown a positive hereditary history in all but three. In four cases, a study of the calcium and magnesium metabolism was made and there was found to be an excessive excretion of the alkali earths. No evidence of infectious origin was found.

Maynard and Scott [146] report sixteen cases occurring in four generations of one family. They conclude that the condition is hereditary and that no infectious cause is suggested.

DISLOCATIONS

Recurrent Dislocation of the Shoulder.—Sever [147] states that the portion of the capsule between the insertion of the triceps on the lower edge of the glenoid fossa and the subcapsularis above is the portion most frequently torn in dislocations of the shoulder. A weakness in the capsule in this region results, and if an atrophy of the triceps and subscapularis occurs, these muscles having a direct action in retaining the head in the glenoid, we have an ideal condition for recurring dislocation. The head usually slips out as the arm is elevated and abducted, and its exciting cause, Sever thinks, is a forward pull of the lower portion of the pectoralis major. For treatment of this annoying condition, the author advises a tenotomy of the pectoralis major, without subsequent suture, and a shortening of the tendon of the subscapularis tendon. He considers capsulorrhaphy of secondary importance. Sever says he has completely divided the pectoralis major

144. Merrill: Am. J. Roentgenol. **7**:480 (Oct.) 1920.

145. Honeij, J. A.: Study of Multiple Cartilaginous Exostosis, Arch. Int. Med. **25**:584 (June) 1920.

146. Maynard, H. H., and Scott, C. R.: Hereditary Multiple Cartilaginous Exostosis, J. A. M. A. **76**:579 (Feb. 26) 1921.

147. Sever, J. W.: Recurrent Dislocation of Shoulder Joint, J. A. M. A. **76**:925 (April 2) 1921.

in about forty cases, with no untoward results, it being possible to adduct the arm quite as well as before; and no essential loss of function has resulted.

Thomas [148] reports his results in twenty-six cases of recurrent dislocation of the shoulder, making a total of forty-four, with his previous report of eighteen. His operation, it will be remembered, consists in reefing the capsule at its inferior portion. Of twenty-four non-epileptic patients, eighteen have shown no recurrences, and four others, on whom more than one operation was performed, may be classed as cured, making a total of twenty-two successful results out of twenty-four. Eighteen shoulders of epileptic patients were operated on and seventeen capsule operations were performed, with a successful outcome in eleven. Thomas says that the approach to the inferior portion of the capsule by the anterior axillary or subpectoral route is very difficult, and he advises the posterior route as the route of approach.

Sandes [149] has devised and successfully performed, in five cases, an operation for control of recurrent dislocation of the shoulder. An anterior incision in the pectoral deltoid sulcus is made and the capsule defined. A strip of fascia lata, 30 by 4 cm., is removed from the thigh, and after being rolled up into a band, is passed through a drill hole made in the head of the humerus, horizontally backward, to emerge through the posterior portion of the greater tuberosity. The band is burrowed through the deltoid, brought up over the clavicle and sewed taut to its other end, while the arm is held abducted 45 degrees. This ligament suspends the humerus approximately through its axis of rotation in abduction to a fixed osseous buttress above. The arm is supported in a sling and rested for six weeks.

Gibson [150] reports seven patients successfully relieved of recurrent dislocation of the shoulder, on whom he performed the Clairmont-Erlich operation. The operation consists of employing a portion of the posterior part of the deltoid muscle as a sling about the neck of the humerus to prevent the usual downward dislocation. The longest time which has elapsed since these operations is more than two years.

Subluxation of the Head of the Radius.—Grossman [151] considers this accident quite common and often overlooked, or else diagnosed as a sprain of the shoulder or wrist. It occurs only in children less than 5 years of age, and is almost always the result of pulling or lifting the child by the wrist. It has been called "traumatic palsy" because of the apparent flaccid paralysis, due to the extreme relaxation of the muscles,

148. Thomas: Surg., Gynec. & Obst. **32**:291 (April) 1921.
149. Sandes: South African M. Rec. **19**:27 (Jan. 22) 1921.
150. Gibson: Canad. M. A. J. **11**:194 (March) 1921.
151. Grossman: Med. Rec. **98**:981 (Dec. 11) 1920.

in order to relieve pain. The head of the radius has slipped out of the orbicular ligament, and it may usually be replaced with a "click" by extreme forcible supination. This is the only treatment necessary. The article is based on a study of thirty-seven cases.

Dislocation of Carpal Bones.—Coues [152] reports a case of dislocation of the carpal scaphoid with partial luxation of the semilunar. The patient was seen several weeks after injury, and excision was advised but refused. There was marked disability of the wrist and thumb. Two other cases of dislocation of the carpal scaphoid associated with luxation of the semilunar were found in the literature. In both of these the bones were excised, with great improvement in function. Honigschmied's experiments on the cadaver lead Coues to believe that intercarpal ligamentous injury with lateral torsion of the wrist would best account for this rather unusual lesion.

Stern,[153] writing on dislocation of the semilunar bone, considers the condition not infrequent. The dislocation is susceptible of reduction in the recent cases by overextension and the use of the Thomas wrench, under complete anesthesia, the bone being held in place by fixation in extreme flexion. Old intractable dislocations should be excised.

Traumatic Dislocation of the Knee Joint.—Platt [154] reports a carefully followed case of true complete traumatic dislocation of the knee joint in a man of 47. Reduction was accomplished and the knee immobilized in plaster for four months, and by a walking caliper for the six weeks following. The patient returned to work in seven months with no instability of the knee, but with flexion limited to 20 degrees. He was seen four years later, after he had served two years in the army. At the time he had from 100 to 110 degrees of flexion and a sound and stable knee. Platt suggests that prolonged immobilization should bring about the same happy result in fresh cases of rupture of both crucial ligaments.

Pathologic Dislocation of the Hip.—Flament [155] calls attention to the not uncommon pathologic dislocations of the hip which occur during or following certain infectious diseases. He has collected eighty cases and finds the relative frequency in the different diseases as follows: typhoid fever, thirty-two; acute articular rheumatism, twenty-four; scarlet fever, thirteen; smallpox, three; Neisser infection, three; influenza, three; erysipelas, one; empyemia, one.

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152. Coues: Boston M. & S. J. **183**:260 (Aug. 26) 1920.

153. Stern, W. G.: Dislocations of Carpal Semilunar Bone, J. A. M. A. **75**: 1389 (Nov. 20) 1920.

154. Platt: Brit. J. Surg. **8**:190 (Oct.) 1920.

155. Flament: Rev. d'orthop. (July) 1920.

FRACTURES

Results in the Treatment of Simple Fractures.—Estes [156] reviews the statistics which he collected as chairman of the committee on fractures of the American Surgical Association. Of the 739 cases analyzed, 120 only were the result of direct violence, and 619 the result of indirect violence. In the British and American groups, the incidence as to age varied considerably, the British showing more than twice as many cases in patients less than 10 years as in any other decade, while the decade of greatest incidence in the American reports was from 20 to 30. In comparing the operative with the nonoperative results, Estes finds that more perfect anatomic alinement was secured by operation, but that the functional recoveries were higher in the cases of patients who were not operated on. The average length of the period of disability is also interesting. For the shaft of the humerus, the period was fourteen weeks; for the head and neck of the humerus, eleven and one half weeks; for the condyles of the humerus, nine weeks; for the shafts of both bones of the forearm, ten and four fifths weeks; for the femur (all sites), seven and thirty-seven hundredths months; for the fractures of the lower leg (all sites), four and three-fourths months.

A Study of Bone Grafting Operations in U. S. Army Hospitals.—Walker [157] has compiled some incomplete but suggestive statistics from the records of the Surgeon-General's office. There were about 25,000 fractures as a result of war wounds in the A. E. F., and 15,165 of these were of the long bones. There have been reported thus far 906 cases of nonunion, and of these, 611 were treated by bone grafting operations. Walker concludes from a study of the results as reported that the autogenous grafts from the tibia have given the best results, and that it is reasonably safe to perform this operation four months after complete healing has taken place. In the ratings for disability which have thus far been made, about one half of the cases (48 per cent.) have shown less than 25 per cent., and about one-fourth (22 per cent.) more than 35 per cent., and 8 per cent. more than 50 per cent. of disability.

Union of Septic Compound Fractures.—Gallie [158] maintains that sepsis favors union in compound fractures rather than prevents it. His investigations have convinced him that inflammation, either caused by infection or resulting from severe trauma with comminution, increases the amount of callus. Bone infection may interfere with union when the bone ends are necrotic. Gallie does not approve of

156. Estes: J. M. Soc. New Jersey **17**:291 (Sept.) 1920.
157. Walker: Ann. Surg. **73**:1 (Jan.) 1921.
158. Gallie: Canad. M. A. J. **10**:407 (May) 1920.

the commonly employed methods of removing sequestrums, cleaning up the sepsis, and allowing the wound to heal and remain healed for several months before any attempt is made to overcome the pseudarthrosis. The method which he has employed in Toronto in the large number of war cases, and of which he speaks most enthusiastically in light of the results, was to remove the sequestrums and at the same time to shorten the limb by approximating the freshened fragments after excising all the scar tissue, suturing the ends with kangaroo tendon. Bony deformities resulting from malunion were corrected by osteotomy at the same time that the patient was operated on for the coexisting osteomyelitis. He points to the great saving of time which this method accomplishes, and says that union practically always occurs.

Schrock [159] believes that after an intra-articular fracture has been reduced with or without operation, the muscles about a joint should be stimulated to active contraction by the second or third day, while the limb is in a splint. He also believes that slight active (never passive) motion of from 5 to 10 degrees should be encouraged, if painless, on the fourth or fifth day, and that on the tenth day, active motion up to half normal should be the goal, increasing to 90 per cent. of the normal range by the third week. No passive motion is to be given and no pain is to be caused.

Compression Fractures of the Lower End of the Radius.—Stevens [160] advocates giving up the name Colles' fracture, and calling the common fracture of the lower end of the radius "compression" fracture. He thinks that there is little actual impaction in most cases; the jamming together of the two portions causes an actual loss of substance; and while deformity can be corrected, it is often impossible to secure absolute anatomic reposition. He urges the importance of early motion, getting rid of all splints, and using a wrist strap only at the end of two weeks. He presents records of four cases.

Troell [161] also concludes that the majority of radial lower end fractures are compression fractures, or are due to hyperextension of the wrist.

Fracture of Tip of Distal Phalanges of Fingers.—Maddren,[162] on the basis of twenty-seven cases, calls attention to the fact that fracture of the tip of the distal phalanx is not a trivial injury. The roentgen ray alone often makes the diagnosis possible. The first symptoms are often mild; but, of twenty-seven patients, one lost 196 days from

159. Schrock, R. O.: Early Active Motion in Intra-Articular Fractures, J. A. M. A. **75**:1320 (Nov. 13) 1920.

160. Stevens: Ann. Surg. **71**:594 (May) 1920.

161. Troell: Ann. Surg. **72**:428 (Oct.) 1920.

162. Maddren, R. F.: Fracture of Tip of Distal Phalanx, J. A. M. A. **75**: 1198 (Oct. 30) 1920.

work, and seventeen others lost an average of sixty days each. The reason for this disablement, in 66 per cent. of the cases, was persistent tenderness and late infection. Early recourse to surgery is indicated. As soon as the diagnosis can be made, an incision should be made into the connective tissue sac about the fracture, to relieve pressure. The route under the nail or a lateral incision may be employed, deep enough to open this sac. The results in his twenty-seven consecutive and unselected cases have been excellent, disability averaging only six and three-tenths days. No man was incapacitated more than thirty days.

Fractures of the Femur.—Speed's [163] excellent article on fractures of the femur analyzes 526 cases. About half were fracture of the neck or through the trochanters, and about half fractures of the shaft. There were 56 deaths, a mortality of 11 per cent. Half of these deaths were due to pneumonia. Intertrochanteric fractures were a little more common than fractures of the neck. Fifty-one operations were performed, with a mortality of 8 per cent. He advocates the abduction treatment in all cases of neck and intertrochanteric cases. He considers that alinement is the most difficult in cases of subtrochanteric fracture, because of the flexion and abduction of the proximal fragment. It is necessary to treat this type by a combination of traction, flexion and abduction of the distal fragment. In fracture of the shaft, after union has been secured by reduction and maintenance in the Thomas or some other splint, or by direct skeletal traction, a walking caliper should be applied before the patient is allowed to walk.

Campbell,[164] in ununited fractures of the neck of the femur, has made a trial of numerous methods. In seven cases, a wire nail was driven through the trochanter, neck and head. In two patients the results were successful, one was improved, and in four the operation was a failure. In twenty cases, tibial grafts were employed, with successful results in fourteen, improvement in one, and two failures. In three cases the results are unknown. In two, the head of the femur was excised and the trochanter placed in the acetabulum after the method of Brackett, with good results.

Fractured Patella.—Hertzler [165] has employed with satisfaction, in transverse fracture of the patella, a method attributed to Dr. G. A. Nickelson of Plains, Kansas. Two stout steel pins are put through the infrapatellar and suprapatellar tendons, as close as possible to the bone fragments, and these projecting ends are approximated by bandages or adhesive plaster. The author presents the roentgenograms

163. Speed, K.: Treatment of Fracture of Femur, Arch. Surg. **2**:45 (Jan.) 1921.

164. Campbell: Southern M. J. **13**:585 (Aug.) 1920.

165. Hertzler: Surg., Gynec. & Obst. **32**:273 (March) 1921.

in a case of transverse fracture with considerable separation of the fragments, in which good apposition and bony union resulted. These pins were not painful to the patient. The leg was supported in a splint, but this was removed from time to time, and the knee exercised. The joint is not opened by this method.

Fractures of the Tibial Spines.—Jones [166] reviews the literature of this rather rare injury, sustained from a severe trauma, acting directly on the knee joint. When the injury is acute, there may be crepitus, tenderness over the inner side of the knee, and locking. The treatment consists of manipulation in order to gain complete extension, and immobilization in plaster for from two to three months.

Stave Fracture of the First Metatarsal Bones.—Cooperman [167] reports a case of stave fracture of the first metatarsal and compares it with the stave fracture of the first metacarpal (Bennett's), which penetrates into the joint between the trapezium and the metacarpal, owing to force acting on the end of the thumb. In the stave fracture of the foot, the fracture penetrates into the joint with the internal cuneiform, but is due to a force exerted on the heel while the first metatarsal head is fixed by weight bearing.

ROENTGENOLOGY

Angle of Epiphyseal Line Between Head and Neck of Femur.—Cramer [168] finds a very wide variation in the angle which the epiphyseal line between the head and neck of the femur makes with the horizontal line. He has examined, roentgenologically, sixty-eight children from 1 to 17 years old. The angle varies from 0 to 50 degrees. Fifty per cent. of the cases showed an angle of approximately 15 degrees.

Bucky Diaphragm.—Potter [169] calls attention to the greatly improved roentgenograms of the spine, which are now possible by the use of the Bucky diaphragm, and to their enhanced value as diagnostic aids. They not only give a heightened contrast between bone and soft parts, but also a greater clearness of outline in the more delicate bony framework of the spine.

[ED. NOTE.—We would go so far as to say that the use of this apparatus has become a sine qua non if we desire to obtain from roentgenograms the greatest amount of information concerning deep bone structures. Its principle is the construction of a parallel grid which moves between the patient and the tube during an exposure, absorbing most of the fogging secondary rays, its own shadow

166. Jones: Colorado Med. **17**:217 (Aug.) 1920.
167. Cooperman: Ann. Surg. **73**:215 (Feb.) 1921.
168. Cramer: Ztschr. f. orthop. Chir. **40**:366, 1920.
169. Potter: Am. J. Roentgenol. **8**:61 (Feb.) 1921.

neutralized by its movement. Plates of the spine taken by this method are in a distinct class by themselves when compared with plates taken by the old technic.]

Diagnosis of Syphilis in the Fetus.—Shipley, Pearson, Seech and Greene [170] have examined, by roentgenograms, about 300 white fetuses, varying from the age of 6 months to, nearly, term, with the purpose of studying the variations from normal. They make the surprising statement that of the first 100 plates studied, fifteen revealed evidence of advanced syphilitic osteochondritis. Ten gave signs of less marked syphilitic disease, and thirty-one revealed one or more bones which varied from the normal and were noted as suspicious. Briefly, 25 per cent. revealed, according to these investigators, marked skeletal signs of syphilis. Practically all the bones may be affected by the syphilitic process; but, even in advanced cases, it seems to interfere very little with skeletal growth.

RESEARCH

Compression Trauma as an Entity.—Nathan [171] has carried out a series of experiments on animals to determine the morbid conditions which ensue in those subjected to different kinds of contusion injuries. He believes these conditions may be expected after fractures. They are twofold: (1) the direct injury to the tissues at the time the force is applied, and (2) the secondary conditions brought about by the tension within the tissue, resulting from the extravasation of blood and the inflammatory exudate. The muscle and nerve fibers, with their end organs, are exceedingly sensitive to compression and irritation, and these secondary conditions are quite likely to lead to disabilities quite out of proportion to the damage from the original compressive force. In the main they consist of: (1) more or less marked and persistent neuromuscular, neurotrophic, and vasomotor disturbances; (2) inhibition of joint motion, with signs of intra-articular injury and intro-articular and peri-articular inflammation; (3) inhibition of joint motion, with the signs of intra-articular inflammation appearing when attempt is made to reestablish joint mobility.

Immunologic Distinctions Between Encephalitis and Poliomyelitis.—Amoss [172] believes that these two diseases can be distinguished by the power of the blood serum of convalescent cases of poliomyelitis to neutralize the virus of poliomyelitis; whereas the blood of convalescent patients in cases of epidemic encephalitis is devoid of this power. He believes that, at the present time, the evidence points toward these two

170. Shipley, Pearson, Seech and Greene: Bull. Johns Hopkins Hosp. **32**: 75 (March) 1921.

171. Nathan: Surg., Gynec. & Obst. **32**:62 (Jan.) 1921.

172. Amoss: J. Exper. M. **23**:187 (Feb.) 1921.

conditions being integrally distinct affections. They both may be infections, the main lesions of poliomyelitis being present in the spinal cord, while those of encephalitis are found in the midbrain.

Strength of Nerve Suture.—Miller [173] has attempted to determine the tensile strength of experimental end-to-end nerve suture, employing the sciatic and popliteal nerves of dogs. These nerves were recovered at varying intervals after suture, and subjected to tests of tensile strength. It was found that this strength was as great after the end of the third week as after the fourth or fifth, being roughly proportionate to the diameter of the nerve. The epineural sutures of silk or catgut seem to play little part after the second week. The investigator advises waiting, in human cases after suture of the sciatic, from six to eight weeks before putting strain on the suture line. In the median and the ulnar, extension of the forearm should be delayed until four weeks have elapsed.

Infection of Tubercle Bacilli into the Metaphyses of Long Bones.—Oliver,[174] discussing Ely's claim that peculiarities in the structure of the bone marrow determine the localization of tuberculous infection in long bones in the epiphysis and metaphysis, states that this is in contrast to the more diffuse process seen so often in the pyogenic infections. Lexer's theory has been that the embolic deposit of bacteria is favored by the anatomic arrangement of the arteries. Oliver's experiments seem to confirm the view that the common localization of the tuberculous infection in the metaphysis and epiphysis is due to the peculiarities of structure of the bone marrow. This tissue in the metaphysis, in contrast to the fatty marrow of the diaphysis, is rich in cells which are peculiarly concerned in the reaction to infection by the tubercle bacilli.

Regeneration of Long Bones.—Martin's [175] experiments on dogs seem to show that regeneration of bone can proceed from the endosteum alone. If there is destruction of the periosteum and cortex, regeneration will, however, not be complete and eventual pseudarthrosis results. The periosteum alone cannot fill the defect as well as the endosteum. The endosteum lining the haversian canals plays a distinct part in the regenerative process. Further experiments by Martin have shown a great difference in the power of the periosteum to produce new bone in growing and adult animals. It is only in the growing dogs that the periosteum seems able to produce callus without the help of the cortex and marrow. If a circular resection of the periosteum and cortex is performed, a pseudarthrosis results. A callus was formed, but a gap

173. Miller, E. M.: Nerve Suture: An Experimental Study to Determine the Strength of Suture Line, Arch. Surg. **2**:167 (Jan.) 1921.

174. Oliver: J. Exper. M. **32**:153 (Aug. 8) 1920.

175. Martin: Arch. f. klin. Chir. **113**:1, 1920; **114**:668, 1920.

persisted between the fragments. The surrounding bone tissue showed a regressive change, and the interesting observation was made that a similar regressive metamorphosis occurred in the parallel bone of the forearm or leg and may proceed to actual pseudarthritis at the same level as on the bone operated on.

Rôle of Cancellous Tissue in Bone Healing.—Todd,[176] after studying cases of chronic osteomyelitis, believes that cancellous bone plays such an important part in regeneration that its integrity ought to be carefully preserved whenever possible. Its activity as a regenerating structure is greatest as the middle of the shaft is approached. Todd thinks we should be most careful in our "disinfection" of cavities, as this procedure often kills the remaining tissue, from which regeneration must be expected.

Autogenous and Homogenous Transplants of Bone in Dogs.—Brooks and Hudson [177] have found that a defect in the shaft of a bone in a dog may be permanently regenerated by a bone transplant removed from another dog. These homogenous transplants were successful in 76.8 per cent. of the experiments, while autogenous transplants succeeded in 84.8 per cent. of the cases.

Recognition of Dead Bone.—Phemister [178] has made physiologic and roentgen-ray studies, attempting to discover characteristics by which dead bone may be recognized in the living body. In the main the characteristics are: 1. Color. Dead bone becomes eventually white instead of purplish, but this whiteness is not an early change; moreover, spongy bone may retain old blood or granulation tissue and appear dark until washed. 2. Erosion. If the dead bone is in contact with an involucrum, either from the inside or outside, its surface may become eroded. 3. Density. This is usually greater than that of living bone, but often deceptive in the roentgenogram if erosion has occurred and a thin flake is roentgenographed on the flat. 4. Line of demarcation. This can usually be detected by the roentgen ray, or at operation, and one of the helpful signs is the preservation, on the dead bone, of the original sharp points or jagged line of fracture. 5. Sinuses lead down to dead bone.

APPARATUS

Fracture Band.—Collins,[179] striving to avoid the ringlike constriction of the bone which the simple Parham band exerts and which, in certain instances, seems to cause bone atrophy, has devised a band

176. Todd: Ann. Surg. **72**:452 (Oct.) 1920.

177. Brooks, B., and Hudson, W. A.: Studies in Bone Transplantation, Arch. Surg. **1**:284 (Sept.) 1920.

178. Phemister: Ann. Surg. **72**:466 (Oct.) 1920.

179. Collins, A. W.: Fracture Reduction and Fixation with a Specially Designed Band, Arch. Surg. **2**:354 (March) 1921.

applied in the same way as the Parham, but shaped like a fenestrated square at one end. This distributes the pressure and avoids the ring-like constriction. He reports its use in a hundred cases, with eight removals.

Protractor.—Robinson,[180] of Pittsburgh, like his fellow townsman Silver, has devised a protractor for measuring the range of joint motion. Silver's pattern (unpublished) is the better, since one may adjust the instrument to measure from the center of joint motion, while the protractor is stable. Robinson's is, however, very convenient, and of vest pocket size and entirely practical.

[ED. NOTE.—We cannot urge too strongly the use of some accurate recorder of joint motion and angles of joint or bone deformity. The experiment of asking any group of well-trained surgeons to estimate by sight alone the range of any given motion or the angle of any deformity may be tried. It will be found that these estimates of sight will vary from 10 to 45 degrees. If records which purport to record progress or retrogression are to be kept, or the patient is to be stimulated to an effort to increase his mobility, a protractor of some sort is absolutely essential. No surgeon can possibly keep accurate records without one.]

MISCELLANEOUS

Adventitious Ligaments Simulating Cervical Ribs.—Law,[181] on operative exploration of four cases of arm pain in which a short or rudimentary cervical rib was present, has found adventitious ligaments stretching from their forward ends to the first rib or sternum. In all the four cases there was pinching of the nerves and vessels between this adventitious ligament and the scalenus anticus muscle. The ligaments were all taut and varied in their points of insertion, one entering the scalene tubercle of the first rib with the muscle, another the costo-clavicular ligament, and a third near the head of the clavicle. After operative intervention, the neuralgia persisted for a short time, quickly becoming less severe and finally disappearing.

Bone Typhoid.—Bohmansson [182] cites a case in which multiple foci of bone infection suggesting typhoid lesions led to the diagnosis. There were almost no other suspicious clinical symptoms. Positive cultures were made from a bone focus. The case suggests that the severity of the primary disease is no index to the likelihood of bone lesions in typhoid.

Anatomic Variations in the Lumbar Spine.—Goldthwait,[183] presenting sixteen illustrative roentgenograms, suggests that more frequent

180. Robinson: J. Orthop. Surg. **3**:41 (Feb.) 1921.
181. Law: Ann. Surg. **72**:497 (Oct.) 1920.
182. Bohmansson: Ann. Surg. **72**:486 (Oct.) 1920.
183. Goldthwait: J. Orthop. Surg. **2**:416 (July) 1920.

roentgenograms of the dorsolumbar region will probably reveal, if properly interpreted, the explanation of many obscure low back and "kidney" pains. We know that many sacral and sciatic pains can be relieved by changing the posture. In the slender, "lanky" individual, with a sagging, visceroptotic posture and a narrowed costosternal angle, the lower ribs may be seen to impinge on a long transverse process. Most cases can be fairly easily relieved when the true mechanical conditions are recognized.

There is a good deal of French literature at present dealing with the abnormalities of the fifth lumbar vertebra as a cause for backache and sciatica. Thus, Nové-Josserand and Rendu [184] report nineteen cases, and conclude that, while the operative removal of a portion of a projecting transverse process has given relief in certain cases, it is usually unnecessary.

Mauclaire [185] has operated on one patient unsuccessfully, but apparently still believes in the operation, suggesting a gallows-shaped incision, vertical in the midline and transverse at the top.

Richards [186] held "sacralization" of the fifth lumbar vertebra responsible for low back pain in 90 per cent. of sixty cases. He thinks that only operative treatment is effective.

[ED. NOTE.—The authors who advocate this serious operation, i. e., the removal of transverse processes, as a routine measure for the relief of symptoms referable to anomalies of the fifth lumbar vertebra seem to us to miss the significance of the fact which, strangely enough, they frequently mention, namely, that the symptoms are often of very recent onset, while the abnormality has, of course, existed since birth. It may be that in certain cases long continued slight irritation may cause hypertrophy of bone and that these overgrowths may cause the nerve-root pressure. As a rule, however, we believe that the two great exciting causes of the symptoms are some sudden back strain or trauma, or the gradual strain, sagging and impingement which an habitually faulty posture brings about. Now, unless actual bony changes in the way of overgrowth have occurred, it has been our experience that conservative treatment by means of the protection afforded by belts or braces and the gradual persistent correction of the posture by means of exercises are quite sufficient to relieve the symptoms. When a strain is superimposed on a vertebral abnormality and unnatural lines of leverage are emphasized by the trauma in this place of lessened resistance, recovery may be more slow than that following a strain of a normal back. A lumbosacral joint of abnormal contour may always be a *locus minoris resistentiae,* but not necessarily one which demands a serious operation.

184. Nové-Josserand and Rendu: Presse méd. **28**:514 (July 28) 1920.
185. Mauclaire: Paris méd. **11**:81 (Jan. 29) 1921.
186. Richards: Bull. et mém. Soc. méd. de hôp. de Paris, July 30, 1920.

Pelvic Articulations During Pregnancy.—Lynch [187] has made a roentgenologic study of the pelvic articulations during pregnancy and labor and the puerperium. He finds that, while it is comparatively rare for the symphysis pubis to be widened during pregnancy, the sacro iliac spaces are almost constantly widened.

Growth Epiphysitis.—Froelich [188] considers that the growth lesions of the tibial tubercle (Osgood-Schlatter disease), Köhler's disease of the scaphoid, apophysitis of the os calcis, trochanteritis, epicondylitis, epiphysitis of the base of the fifth metacarpal, etc., should all be grouped under the one name of growth epiphysitis. He considers them all mild forms of osteomyelitis, whose importance is their liklihood of being mistaken for tuberculous bone lesions.

[ED. NOTE.—We are in accord with the proposal to group all these conditions, now frequently erroneously designated by the name or names of the men who first described them, under some such name as growth epiphysitis, with the exception of Köhler's disease. This seems to us still to be unexplained in its etiology and not to belong to this large group. Köhler, at the Radiographic Congress at Wiesbaden, in 1908, first described this peculiar lesion of the scaphoid bone of the foot, and Mouchet and Roederer [189] and Regaud [190] recently have added to our knowledge. It is a peculiar change in the structure and apparently represents a developmental abnormality of a nontraumatic and noninfectious origin, since the two bones may be affected at the same time in one person. The bone is narrowed in width, sometimes almost disk-shaped, and of far greater density than normal. We do not agree with Froelich that the probable cause of what he designated growth epiphysitis is a low grade osteomyelitis. It seems to us that it is entirely unnecesary to suppose an infection, both from the speedy relief from protection alone and from the absence of other signs of infection. We consider it most likely to be merely an irritation of a growing center by a strong muscle pull. They all occur in regions in which a powerful muscle is attached to a small ununited center of bone growths. We believe them to be, generally speaking, indirect traumatic inflammations in subjects who have not attained full growth, and in the neighborhood of those epiphyses that are not yet united and subjected to powerful muscle traction.]

Curative Play.—Schauffler [191] describes an excellent curative play system in force at the Mercy Hospital in Kansas City. He is enthusiastic over the results obtained in cases of weakened muscles and stiff

187. Lynch: Surg., Gynec. & Obst. **30**:575 (June) 1920.
188. Froelich: Paris méd. **10**:430 (Dec. 4) 1920.
189. Mouchet and Roederer: Rev. d'orthop., July, 1920.
190. Regaud: J. de radiol. et d'electrol. **4**:453 (Oct.) 1920.
191. Schauffler: J. Orthop. Surg. **3**:98 (March) 1921.